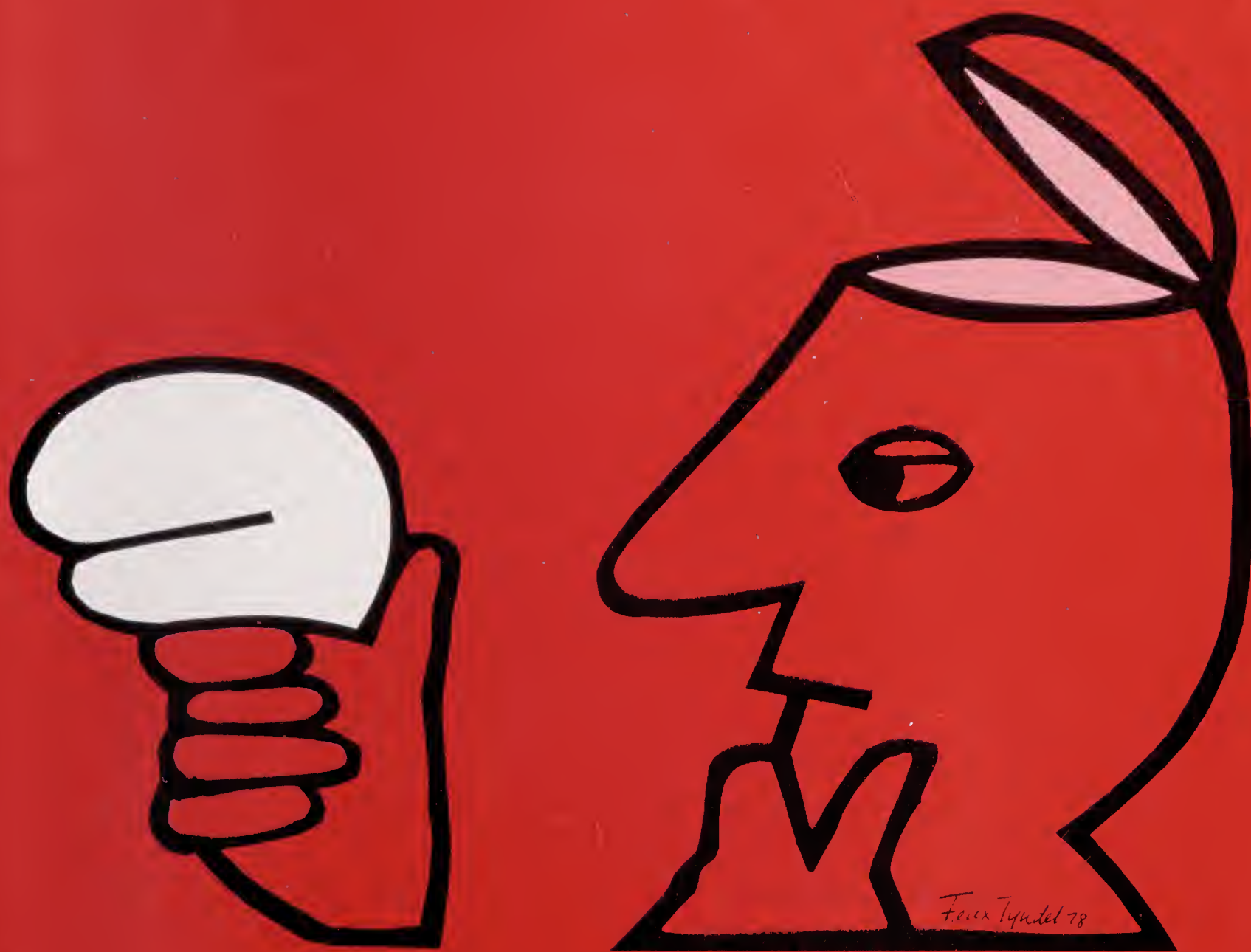


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Established December 1923
An Undergraduate Publication

Volume LVI
Number 3

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GUIDELINES FOR PUBLICATION

- 1) All articles intended for publication should be submitted to the editors before the deadline which is usually six weeks before the release of the Journal
- 2) Submitted articles should be typed (or legibly printed) double-spaced, single-sided on paper 8½" x 11" (letter size) and they should not be more than 15 to 20 pages (3000 to 4000 words)
- 3) Illustrations, graphs, diagrams and figures should be put on a separate sheet and identified by Arabic numerals. A list of explanatory legends for all the figures and diagrams should be put on another sheet. Do not write on the back of photographs
- 4) References should be checked with the original sources, designated by Arabic numerals in the text, numbered in order of appearance and put on a separate sheet. Each periodical reference should include in order the author's name and initials, title of the article, Journal name, volume number, page number and year (eg, 1. Rosen HJ. Lumbar Invertebral disc surgery: review of 300 cases. *Can Med Assoc J.*, 101: 317, 1969)
- 5) The editors reserve the right to make such editorial changes as are deemed necessary to ensure correctness of grammar and spelling, clarification of statements and conformity to Journal style
- 6) Acceptance for publication is subject to the understanding that such material is submitted solely to the Journal and will not be reprinted without the consent of both the editors and author.

Analysing Electrical Signals From The Brain

John Doyle, M.A.Sc., 8T2

Institute of Biomedical Engineering, U. of T.

Computer technology permits precise and objective analysis of EEG signals. The author describes a system which is able to record changes in the frequency and amplitude of signals over prolonged periods of time. This may have clinical application in monitoring recovery from coma, surgery or from cardiac arrest.

The study of the electrical activity of the brain (electroencephalography) has a history of about 50 years, beginning essentially with a description of the human electroencephalogram (EEG) by Berger in 1929¹. An illustration of a typical scalp EEG recording is shown in Figure 1.

As can be seen from Figure 1, the EEG is a low-frequency, low-amplitude electrical signal which takes a somewhat random oscillatory form with a number of rhythms and transient discharges which are now well-known to electroencephalographers. In the absence of deliberate sensory stimuli, such activity is usually referred to as the spontaneous EEG and is divided into frequency bands for descriptive purposes. Best known is the Alpha rhythm (Berger rhythm) whose frequency is in the range of 8-12 Hz. This rhythm is apparent in the last two tracings of Figure 1. Other rhythms also exist: Beta: above 12 Hz; Delta: below 4 Hz; Theta: 4 Hz to 8 Hz.

Despite decades of research, the physiological sources of the EEG are still poorly understood. Although the gross electrical activity measured at the scalp is believed to be due to the activity of neurons (mainly pyramidal cells) in the cerebral cortex, the exact nature of the scalp potentials and their relation to cortical activity remains unclear. A number of ex-

planations, however, have been forwarded³ and in particular, studies by Elul⁴ suggest that "the EEG represents the output of a relatively small group of cortical neurons which are synchronized by subcortical inputs... (however) any given nerve cell does not remain synchronized with other cells—and with the EEG—over any extended period of time".

In spite of these difficulties, a large body of empirical knowledge con-

cerning the EEG exists which makes electroencephalography a particularly useful non-invasive diagnostic tool in neurology. It can only be expected that its usefulness will increase once the basic physiological mechanisms of EEG electrogenesis have been elucidated.

How an EEG is Done

Although the procedure for recording an EEG will vary from department to department, many common

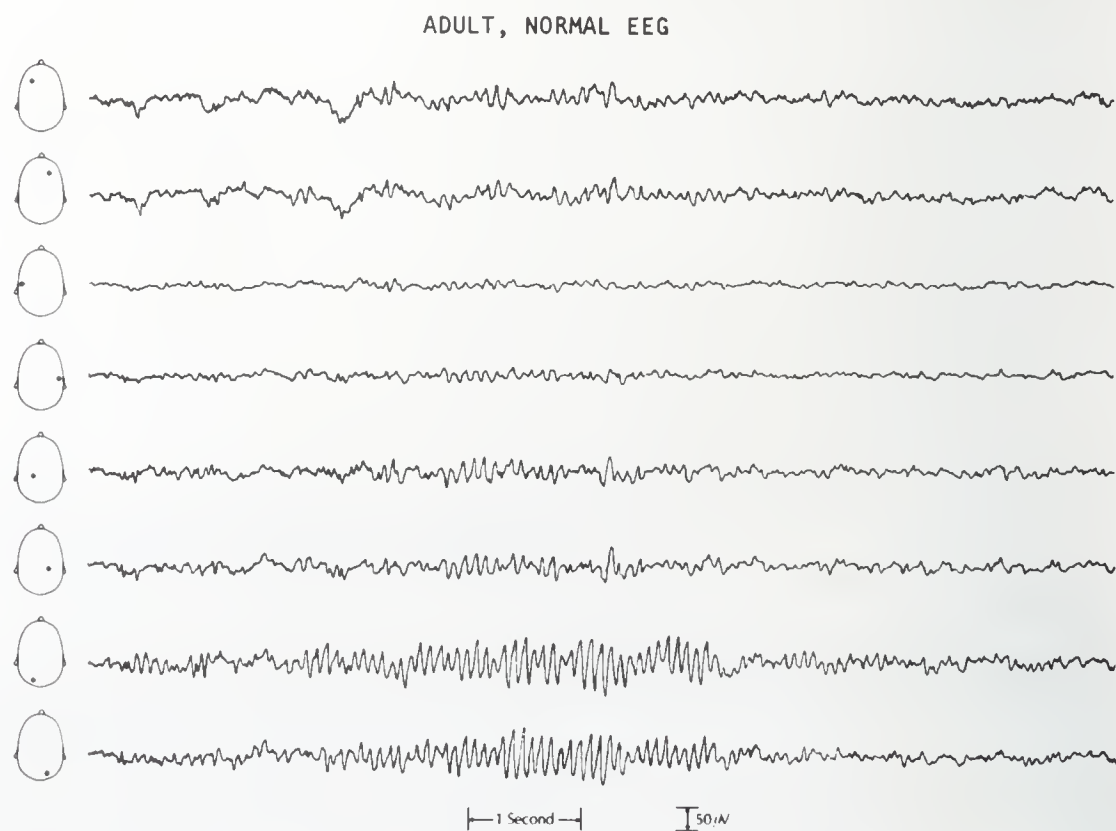


Figure 1: A typical adult EEG recording.² Note the alpha rhythm present in the posterior channels.

elements exist. First, scalp electrodes are mounted at specific sites on the patient's head. The most commonly used sites are those established in the internationally accepted "10-20" system. As can be seen from Figure 2, in this placement system, the median electrodes are distributed at spacings of 10% or 20% of the nasion-inion distance (hence the term "10-20" system).

Usually 8 or 16 channels of EEG signals are displayed at one time. To display the activity at all the electrode sites shown in Figure 2, several runs with different channel selections are necessary.

The electrodes themselves are small gold or silver-silver chloride discs less than a centimeter in diameter. They are mounted on the scalp using conductive electrode paste, gauze and colloidion after the skin has been cleansed and degreased with acetone. Care must be taken that pathogens are not spread from patient to patient via EEG electrodes.

Typically, the EEG is displayed on an EEG machine (electroencephalograph) at a pen sensitivity of 7.5 microvolts/mm and a paper speed of 30 mm/sec. Recent developments in EEG machinery include automatic tape recording of

EEG signals, EEG transmission over telephone lines and ink-jet paper writers, but otherwise, most EEG machines are essentially the same.

A typical EEG recording session takes about one hour, perhaps more if a sleep recording is done. A number of runs with various channel selections is done, typically four or more. On some runs hyperventilation and photic stimulation may be employed in order to bring out possible EEG abnormalities. If a sleep study is desired, the patient may be given a fast-acting barbiturate or chloral hydrate to induce sleep.

Clinical EEG Analysis

Virtually all clinical EEGs are visually examined by electroencephalographers for possible abnormalities. Two kinds of abnormal EEG are shown in Figures 3 and 4. Other kinds of abnormalities found in the EEG may be caused by cerebral tumors, various forms of epilepsy, cerebrovascular accidents, head injury, brain infections, psychiatric disorders and brain death⁵. This explains the clinical usefulness of the EEG.

Analysis of the EEG always begins by considering the physical and

psychological state of the subject being recorded, as well as the recording site on the scalp. For example, the Alpha rhythm is most prominent over the back of the head (occipital region) when the subject is relaxed, inattentive and awake with eyes closed. Under normal conditions this rhythm is never found in the frontal head region, thus introducing one of many rules concerning EEG topography which EEG interpreters must be concerned with.

Besides topographic considerations, electroencephalographers must consider the distribution of EEG signal energy among the various frequency bands, the amplitude of the EEG, the similarity of EEG signals from homologous EEG channels, the existence of possible "spike-like" peaks in the EEG and a myriad of other matters.⁵

Further complicating the situation is the problem of non-neuronal signals (artifacts) contaminating the EEG. For example, swallowing and other movements generate electromyographic potentials which make EEG interpretation impossible. Eyeblinks create "Slow" waves in frontal channels which can be mistaken for pathological slow waves such as might be expected from a

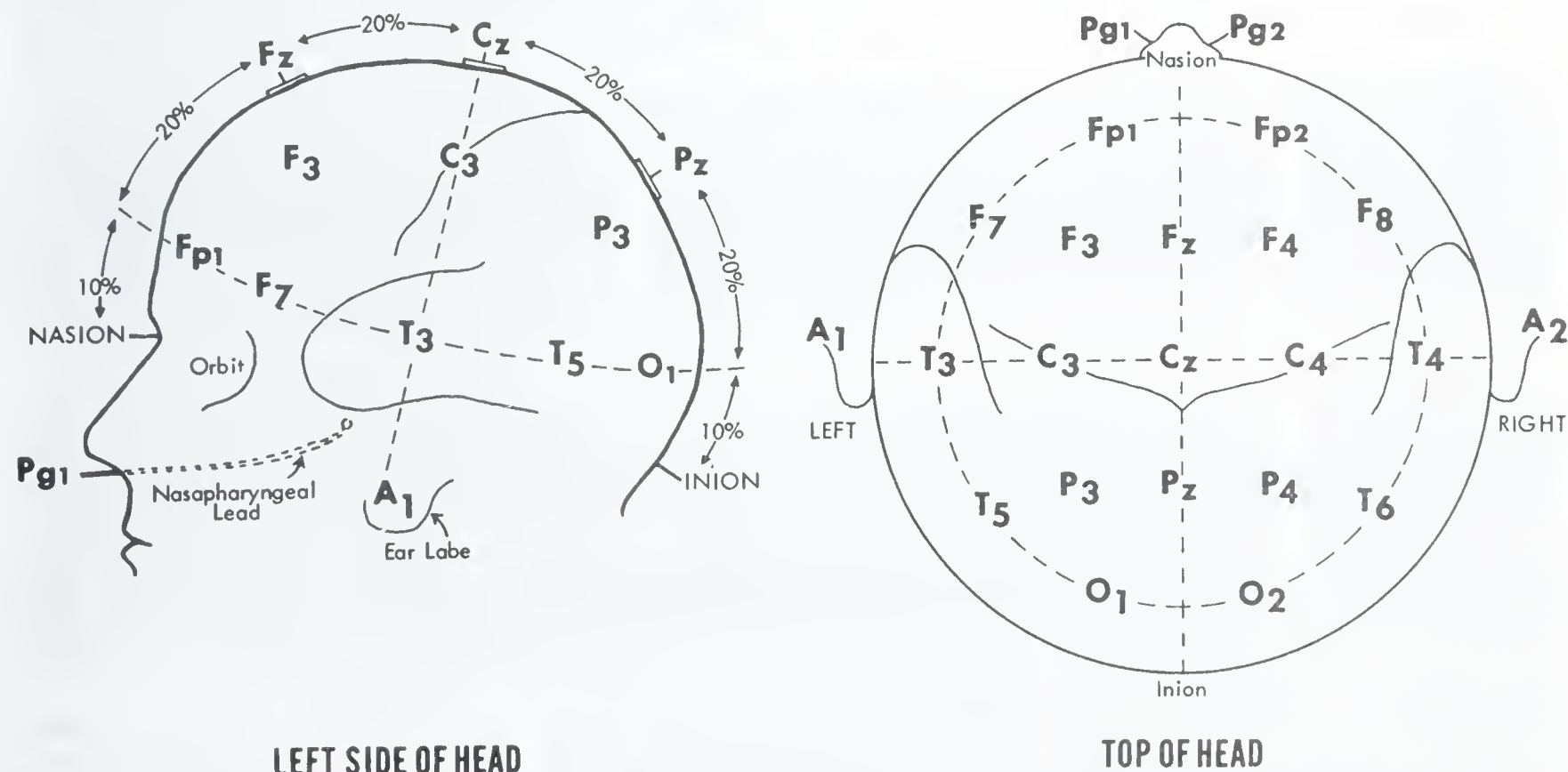


Figure 2: Electrode placement for the internationally accepted "10-20" system of electrode placement. Note the following mnemonics: F = frontal; C = central; P = parietal; O = occipital. Note also that electrodes on the left are odd numbered, while those on the right are even.

ENCEPHALOPATHY (HEPATIC COMA)

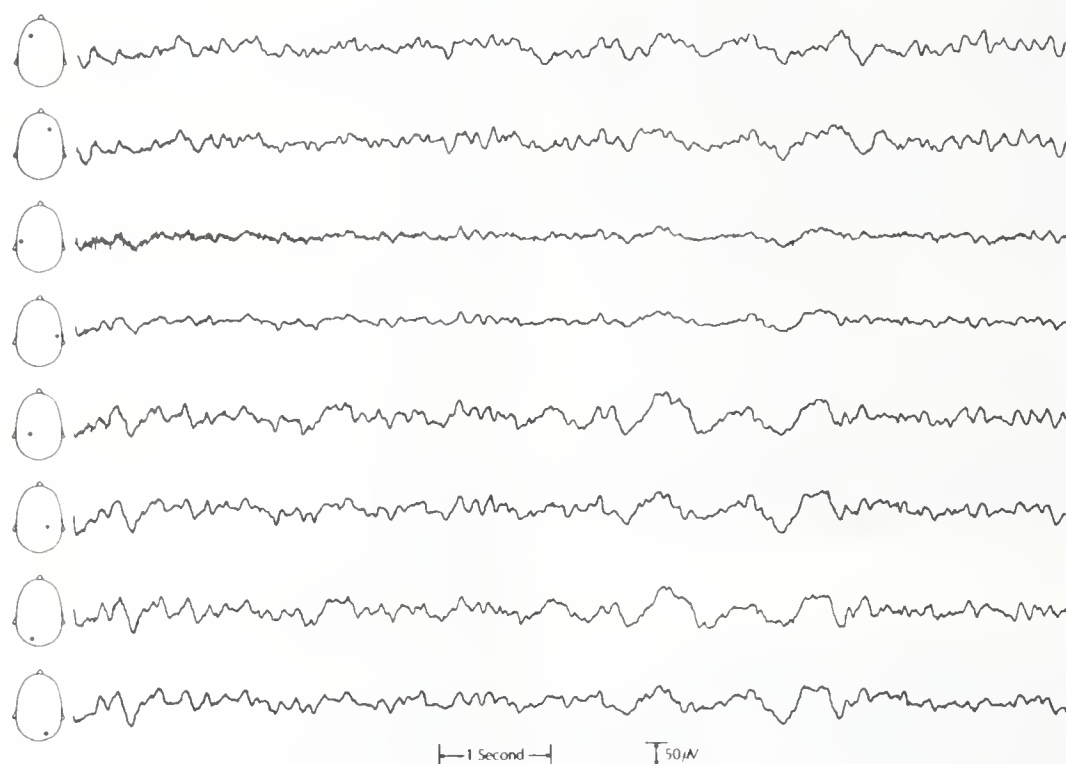


Figure 3: An example of the EEG in hepatic encephalopathy.² Note the large amount of delta activity present.

DRUG INTOXICATION (BARBITURATE)

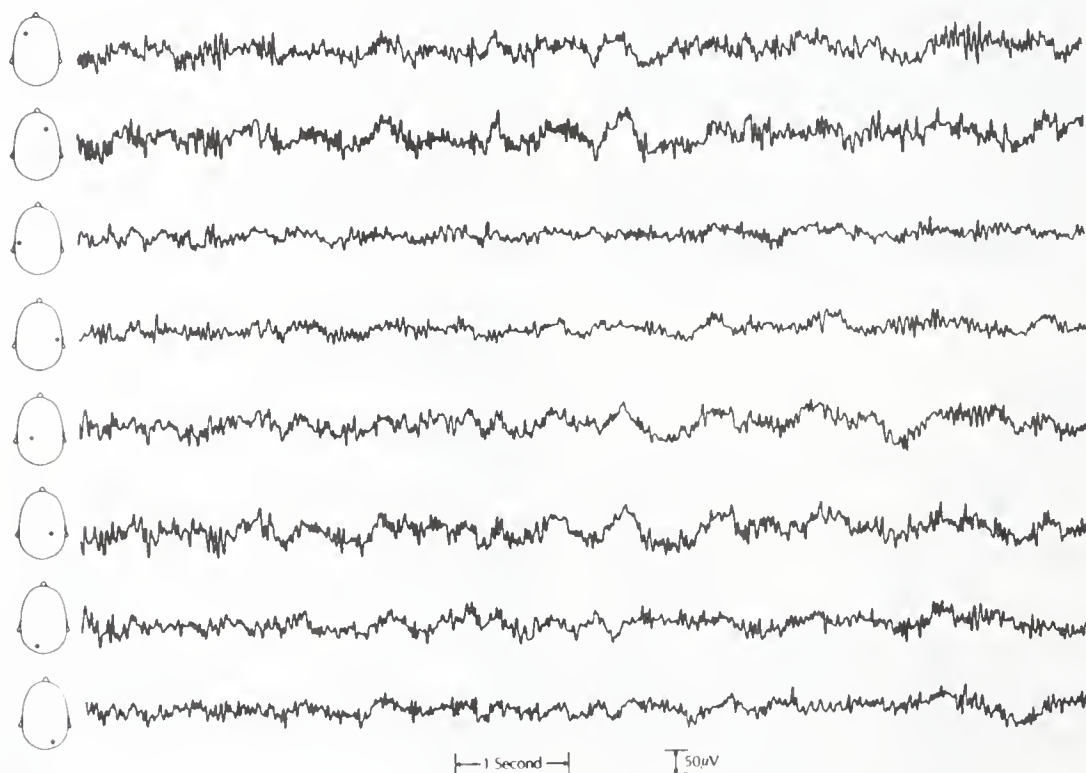


Figure 4: Example of the EEG in barbiturate overdose.² Note the large amount of beta activity present.

cerebral tumor. Poorly operating electrodes may create voltage changes which mimic epileptogenic activity.⁵ The complexity of EEG analysis makes it clearly a task to be attempted only by properly trained specialists. Even then, disagreements regarding interpretation often arise.

EEG Analysis by Computer

The subjective nature of EEG interpretation has been a motivating factor in efforts to develop quantitative EEG descriptions and thereby make EEG interpretation more objective. So far, little of this effort has had any clinical impact, but the rapidly decreasing cost of computer hardware will eventually mean that

some clinical EEGs will routinely be analyzed by computer.

Many kinds of computer analyses of EEG signals are possible. One common type of analysis involves computing the EEG signal energy in the delta, theta, alpha and beta bands. This is an important aspect of visual EEG analysis, but visual analysis alone will not yield this information precisely. Amplitude analysis of the EEG can also be done by computer. Efforts to automatically diagnose clinical EEGs as normal or abnormal have been done. For these and other aspects of computer analysis of the EEG, the interested reader is referred to the review by Cox *et al.*⁶

The author's interest in computer analysis of EEG signals began as a graduate student in electrical engineering in 1972. This interest led to his recent development of a microcomputer-based system for EEG analysis as an aid to brain function monitoring. Unlike a conventional EEG machine, with a paper speed of 30 mm/sec, this system has a paper speed which may be as little as about one thousandth the usual EEG tracing speed. Its intended application is to permit recognition of changes in the EEG over periods of hours or days. Clinical situations where easy recognition of EEG changes corresponding to changes in brain function would be desired include monitoring recovery from coma, surgery or from cardiac arrest.

Figure 5 shows a block diagram for the system. The EEG from the patient is amplified 200,000 times and is bandpass filtered (0.5 to 30 Hz) by an EEG amplifier/filter system. The resulting signal is output directly on Channel One of the chart recorder. Because of the slow speed of the recorder, only the magnitudes of the signal tracings are apparent from the thick band of ink obtained. (Figure 7). This channel of the chart recorder tells us about the EEG amplitude and allows us to recognize some kinds of EEG artifacts.

The Second Channel contains frequency-related information computed by the microcomputer. Specifically, the microcomputer is programmed to calculate and display the interval between changes in the

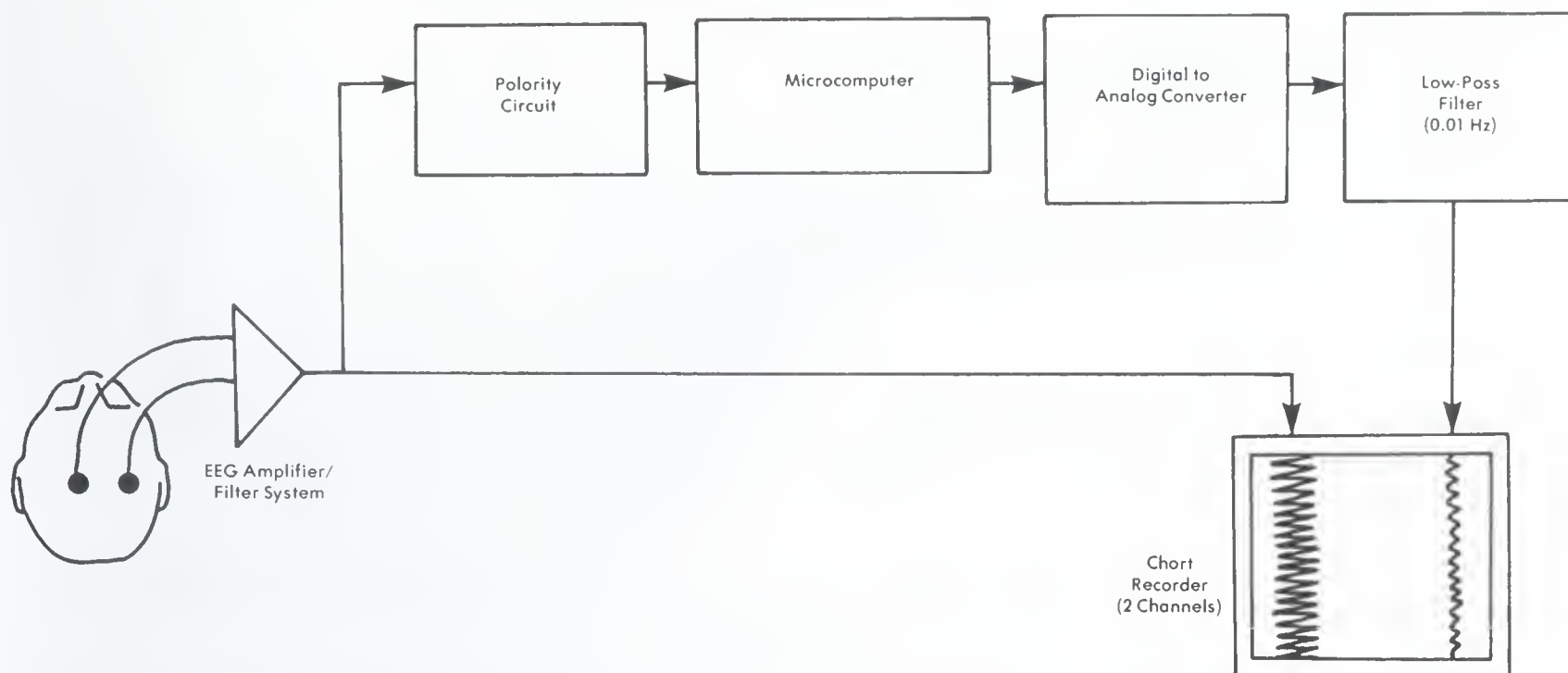


Figure 5: Block diagram of the EEG brain function monitor developed by the author

EEG polarity (sign).

Figure 6 illustrates these intervals for a hypothetical EEG signal. These intervals are also known as “zero-crossing intervals” or “EEG periods”. In clinical electroencephalography these intervals, if desired, are usually calculated by hand, an obviously tedious process.

The zero-crossing intervals deter-

mined by the microcomputer to the EEG shown in the top tracing of Figure 7 are shown in the bottom tracing of the same Figure. The fluctuations are averaged by the low pass filter shown in Figure 5. The effect of this filtering is to decrease the variation of the zero-crossing interval information. This “smoothing” of the tracing facilitates its interpretation,

but responses to sudden EEG changes then take longer to occur.

The subject in Figure 7 is a normal Caucasian female in her twenties. An examination of the lower tracings in Figure 7 (giving the average zero-crossing interval) reveals zero-crossing interval of about 50 milliseconds. This corresponds to an average frequency of about 10 Hz.

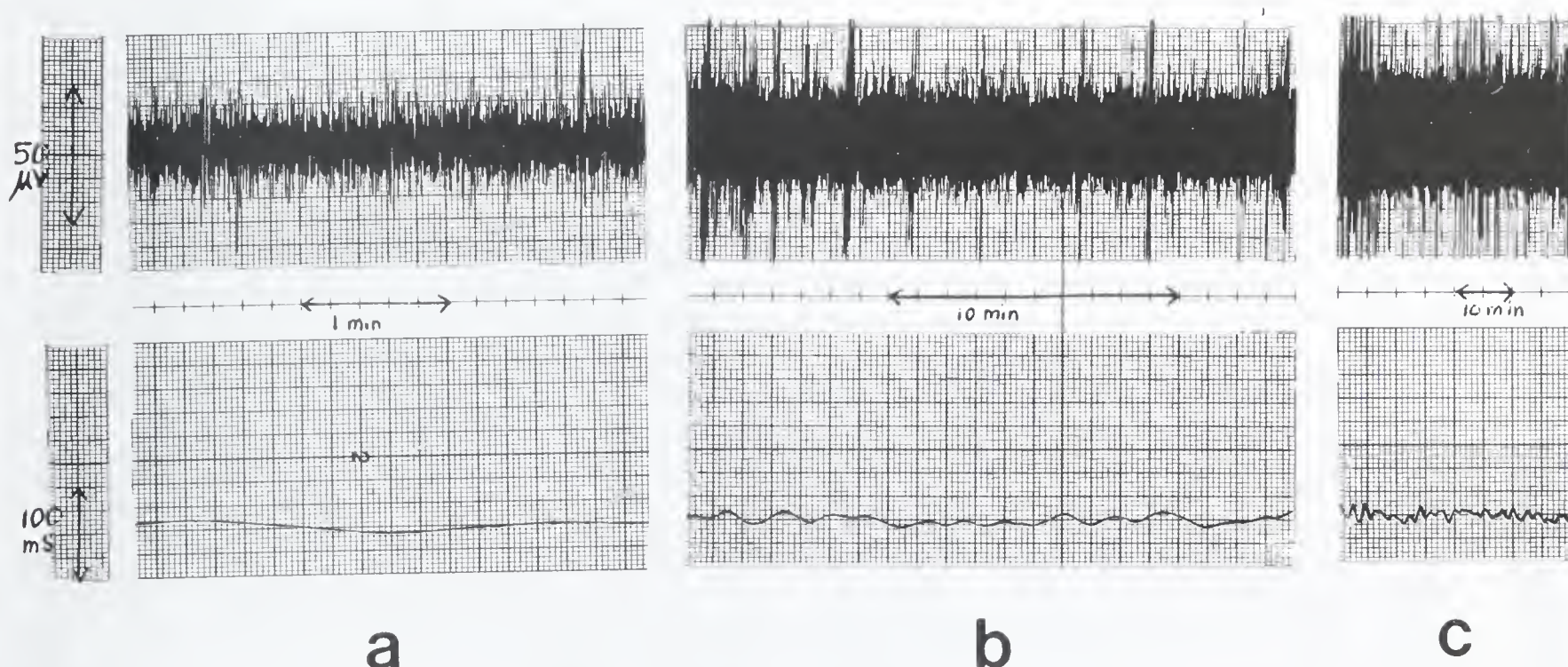


Figure 7: Average zero-crossing intervals for subject F.S. calculated by the system shown in Figure 5. The paper speed shown in (b) would typically be used for surgical monitoring, while the speed shown in (c) would be suitable for monitoring comatose patients.

The Student Aid Committee of the University of Toronto Medical Alumni Association has a budget of \$44,000 for 1978-79. This money is to be spent as follows:

Scholarships (3 year scholarships of \$250 each, and a graduation scholarship of \$600)	\$1,350.
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Undergraduate bursaries (This bursary money is awarded through the Faculty Undergraduate Awards Committee)	11,250
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Graduate bursaries	1,000
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Medical Student Summer Fellowships (12 Fellowships at \$1,500. 2 of these are for Family Practice)	18,000
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Medical Student Transportation Fund (This Fund is primarily intended for Medical student electives, although applications for travel connected with work experience are also accepted.	3,000
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The maximum individual grant is \$150.

Applications for 1979-80 electives are accepted up to April 16, 1979 for the period June 5, 1979 to November 19, 1979, and up to October 1, 1979 for the period November 20, 1979 to May 6, 1980.

The \$3000 is budgeted for the period June 5, 1979 to November 19, 1979. Completed application forms must be delivered in person to the Medical Alumni Association office during regular office hours or postmarked before midnight on the deadline dates. Late applications will NOT be considered.)

Fund for Educational Activities	1,000
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Not yet assigned	2,000
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There are three additional Alumni Association scholarship awards. The *Samuel J. Streight, O.B.E., M.D., Scholarship in Internal Medicine* (value \$240) is awarded annually to a student in the final year who has high standing in Internal Medicine. The *Dr. R.E. Haist Award in Physiology* (value \$100) is awarded annually to a second year student who has achieved high standing in Physiology. The *Dr. K.J.R. Wightman Award in Internal Medicine* (value \$100) is awarded annually to a final year student who has achieved high standing in Internal Medicine.

The Association administers two special funds. The *Dr. Bernard A. Manace Award* (value \$150) provides assistance to a Medical student from any year who is undertaking an elective or summer Medical experience in Israel. Preference is given to students working in the area of Allergy or Immunology. Consideration is given to academic standing. The *Dr. L. Nelles Silverthorne Award* (value \$140) provides assistance to a Medical student from any year who is undertaking an elective or summer Medical experience in a remote area. Preference is given to a student working in a mission

hospital. Consideration is given to financial need. The deadline for applications for these two awards is March 1, 1979 for the subsequent 12 month period. As with the Medical Student Transportation Fund, late applications will not be considered.

The Association has four loan funds. The *Dr. Ernest J. Clifford Medical Alumni Association Undergraduate Loan Fund* provides short term, interest free loans to Medical students who require financial assistance during periods of temporary difficulty. The *Robert Elgin Towle Loan Fund* provides interest free loan assistance to women undergraduates. The *Medical Alumni Association Graduate Loan Fund (including the Emma Bissell and Agnes E. Hamshaw Funds)* provides interest free loans of up to \$1,500 per year to physicians taking postgraduate training. The *Alexander and Martha Dalrymple Loan Fund in Psychiatry* provides interest free loans for physicians taking postgraduate training in Psychiatry. This fund is administered jointly by the Association and the University Department of Psychiatry.

Further information about any of the above, and application forms where required, can be obtained from Mrs. N.S. Shenstone, Secretary, Medical Alumni Association at 280 Bloor Street West, Room 10, phone 921-1150.

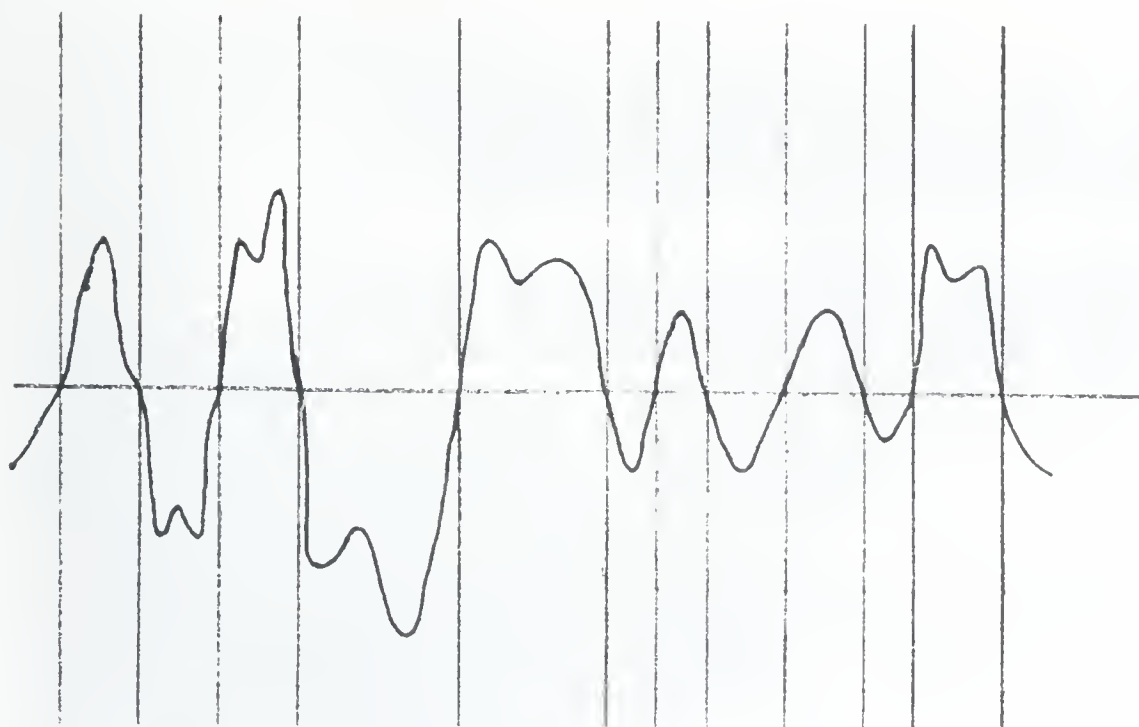


Figure 6: Diagram to illustrate zero-crossing intervals in the EEG.

Note also that the peak-to-peak EEG amplitude is under 50 miliseconds. This corresponds to an average frequency of about 10 Hz. Note also that the peak-to-peak EEG amplitude is under 50 microvolts, and

that apparent band thickening occurs with decreasing paper speed.

Future Work

Preparations are now underway to begin studies to examine this

system's potential as a patient monitor for surgical patients and for patients recovering from coma. It is anticipated that this method of analysis will be a useful adjunct to conventional means of neurological assessment in such patients. □

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Endorphins: A New Frontier

George Yu 8T0

The extract of the opium poppy, *Papaver somniferum*, is among the oldest medicines used by man. The ancient Greeks took it to relieve pain, treat diarrhea and produce euphoria. However, the mechanisms by which the opiates exert their diverse effects remained a mystery through the centuries. It was not until the last few years that scientists managed to gain a better understanding of their mode of actions which consequently led to the discovery of the existence of their counterparts, the endorphins (endogenous morphines), in the human body. Since then, this area of research has expanded into one of the most important fields in all of science and medicine.

The Opiate Receptors

For many years, scientists have postulated that opiates act by binding to specific receptors on the neurons. The reasons for this hypothesis is the high degree of structural and steric specificity which characterizes opiate actions. The opioid effects are produced exclusively by the (-)-isomers and substitution of a side chain may transform the molecule into an opiate antagonist. The existence of extremely potent opiates, such as etorphine which produces analgesia and euphoria in doses as low as 0.1 mg, also lends support to the receptor theory. Opiate receptors have now been demonstrated by the use of radioactive stereospecific opiate ligands.

Search for the Endorphins

The opiate receptors are present only in vertebrates and have highly specific distribution in the central nervous system, especially in the 'pain' pathways. Since vertebrates are not born with morphine in their body, these facts prompted researchers to look for an endogenous opiate ligand—the missing key to the lock. In 1975, Hughes *et al.* identified two opioid pentapeptides from pig brain: methionine-enkephalin and leucine-enkephalin. In the following year Li *et al.* isolated another opioid peptide, B-endorphin, from camel pituitary while Guillemin *et al.* demonstrated the existence of two other polypeptide endorphins, the A- and Y-endorphins, from pig brain. All these endogenous peptides that bind to the opiate receptors are grouped under the generic name of 'endorphin'.

Types of Endorphins

Methionine-enkephalin (H-Tyr-Gly-Gly-Phe-Met-OH) and *Leucine-enkephalin* (H-Tyr-Gly-Gly-Phe-Leu-OH)

These are five amino acid peptides with molecular weights of approximately 800 daltons. On a molar basis, these peptides are about as active as morphine. These endorphins appear to be neurotransmitters for the following reasons:

1) Short half-lives from rapid peptidase deactivation,

- 2) Localization in synaptic terminals,
- 3) Rapid actions by iontophoresis,
- 4) Naloxone (a specific opiate antagonist) blockade of both iontophoretic and synaptic evoked effects,
- 5) Similar distribution of enkephalins and opiate receptors in brain,
- 6) Release into CSF upon electrical stimulation of the brain.

The amino acid sequence for met-enkephalin corresponds to positions 61-65 of B-lipotropin (B-LPH), a 91 amino acid pituitary hormone of ill-defined functions and no opioid activity.

B-endorphin

This is a 31 amino acid peptide with a molecular weight of about 3100 daltons and corresponds to B-LPH₆₁₋₉₁. It also appears that B-LPH is the precursor of B-endorphin. Mains *et al.* have shown in the pituitary that ACTH and B-LPH have a common precursor, and that ACTH, B-LPH and B-endorphin are stored and subsequently released together. In all biological assays tested, B-endorphin is considerably more active on a molar ratio than enkephalin and the duration of activity of B-endorphin is also much longer. This action favours its role as either a hormone or a neuromodulator.

Other Endorphins

Besides B-endorphin, other long chain endorphins such as A-endorphin (B-LPH₆₁₋₇₆), Y-endorphin (B-LPH₆₁₋₇₇) and D-endorphin (B-LPH₆₁₋₈₇) exist in small

amounts in the pituitary. In addition, anodynin, a 600 daltons opioid peptide, has been assayed in human blood sample. These substances probably represent various breakdown products of *B*-endorphin.

The Endorphin Neuronal System

The met- and leu-enkephalins were detected in similar areas in the neuraxis with met-enkephalin having a higher concentration. High concentrations of enkephalins are found in the striatum, hypothalamus, thalamus, hippocampus and the brain stem. The enkephalins are present in small amounts in the cortex and cerebellum and absent in the pituitary. In the spinal cord, enkephalins are detected in the gray matter, especially the substantia gelatinosa. Large amounts of enkephalins are also found in the longitudinal muscle - myenteric plexus layer of the small intestine.

By contrast, *B*-endorphin is mainly localised in the pars distalis and pars intermedia of the pituitary and in the hypothalamus. *B*-LPH has also been detected in the hypothalamus and in smaller amounts in the hippocampus, central gray, thalamus, pons and the spinal cord.

The concentration of *B*-endorphin in the brain is much lower than that of the enkephalins and the amino acid sequence of met-enkephalin is contained in the *B*-endorphin molecule. The above facts raise the question of whether *B*-endorphin in the brain merely exists as a precursor for the enkephalin. However, immunocytochemical studies of the brain indicated that the cell group distribution and synthesis of the enkephalins and *B*-endorphins are quite independent. Two separate endorphin pathways have thus been delineated in the brain—a *B*-endorphin system comprising a single cell group in the hypothalamus with long axons innervating the midbrain and the limbic system and an enkephalin consisting of multiple cell groups throughout the brain and the spinal cord with relatively short axons, Fig. 2.

The actions of the enkephalin as a neurotransmitter have been studied extensively. Generally, enkephalin

inhibits cellular responses except in the hippocampus and the Renshaw cells (excitations in these areas are probably mediated by inhibiting the inhibitory interneurons). These activities are naloxone-reversible, showing that they are probably mediated through opiate receptors. The electrophysiological basis of their action appears to involve the sodium conductance on the pre- and post-synaptic membranes.

Significance of Endorphins

Analgesia

Richardson *et al.* demonstrated that the focal electrical stimulation of the human brain produces naloxone-reversible analgesia with an elevation of enkephalin level in the CSF. Significant analgesia was also seen in rats after sessions of foot-shock stress with accompanying rise in plasma *B*-endorphin level. These combined with the specific distribution of endorphinergic neurons along the paleospinothalamic 'pain' pathway suggest the existence of an 'intrinsic analgesia system' (IAS) which mediates its actions via the endorphins.

The midbrain periaqueductal gray and raphe nucleus are important components of this system. The enkephalins are abundant in the periaqueductal gray interneurons which send their axons to synapse on the raphe nucleus. This, in turn, projects to the spinal trigeminal nucleus and, via the dorsolateral tract, to the dorsal horn 'pain' interneurons. The IAS may act as a negative feedback loop for pain and seems to be activated only during stress or trauma, i.e. as a means of turning off chronic pain. The observations that naloxone accentuates clinical pain in post-surgical state and tooth extraction also lend weight to this hypothesis.

An alternative approach to the study of the IAS is to investigate pathological pain conditions where dysfunction of the system can be anticipated. The measurements of endorphins in CSF of patients with severe chronic organic pain revealed subnormal levels. A likely explanation is the general depression of the IAS in these patients, which

would predict a general hypersensitivity to noxious stimuli a condition already reported by other investigators. However, in patients with psychogenic pain, CSF endorphin levels were supernormal. Since the pain in these patients is a manifestation of psychic disturbance, the elevation of endorphins level may be linked to the psychological stress. The positive correlation between the degree of depression and the endorphins level appears to substantiate the postulate.

Besides the possible change in activity of the IAS under pathological situations, a normal diurnal rhythm of the IAS may exist. This suggestion was alluded to by the observation that a diurnal variation of naloxone-induced hyperalgesia (decrease in pain threshold) occurred in normal mice.

An interesting finding by Levine *et al.* showed that analgesic effect of placebo was naloxone-reversible, indicating that endorphins may be the mediators, which implies that the endorphin system can be triggered by suggestion. Yet the findings of a similar study with low doses of naloxone could demonstrate no naloxone effect and the fact that naloxone failed to modify hypnotic analgesia have vitiated the postulate.

Addiction

The discovery of the antinociceptive actions of the endorphins has posed some hope for a possible non-addictive analgesic. However studies revealed that the endorphins produced tolerance and dependence similar to those of morphine. Furthermore, cross-tolerance between morphine and the endorphins was demonstrated. Like morphine, these chemicals decrease the intracellular concentration of a second messenger, cAMP, in the responsive cells. This process is achieved by inhibiting the activity of the membrane-bound adenylate cyclase which converts ATP to cAMP. A high intracellular concentration of cAMP may lead to the withdrawal syndrome of physical dependence.

Acupuncture

The ancient method of curing disease and relieving pain by acupuncture remains an integral part of traditional Chinese medicine.

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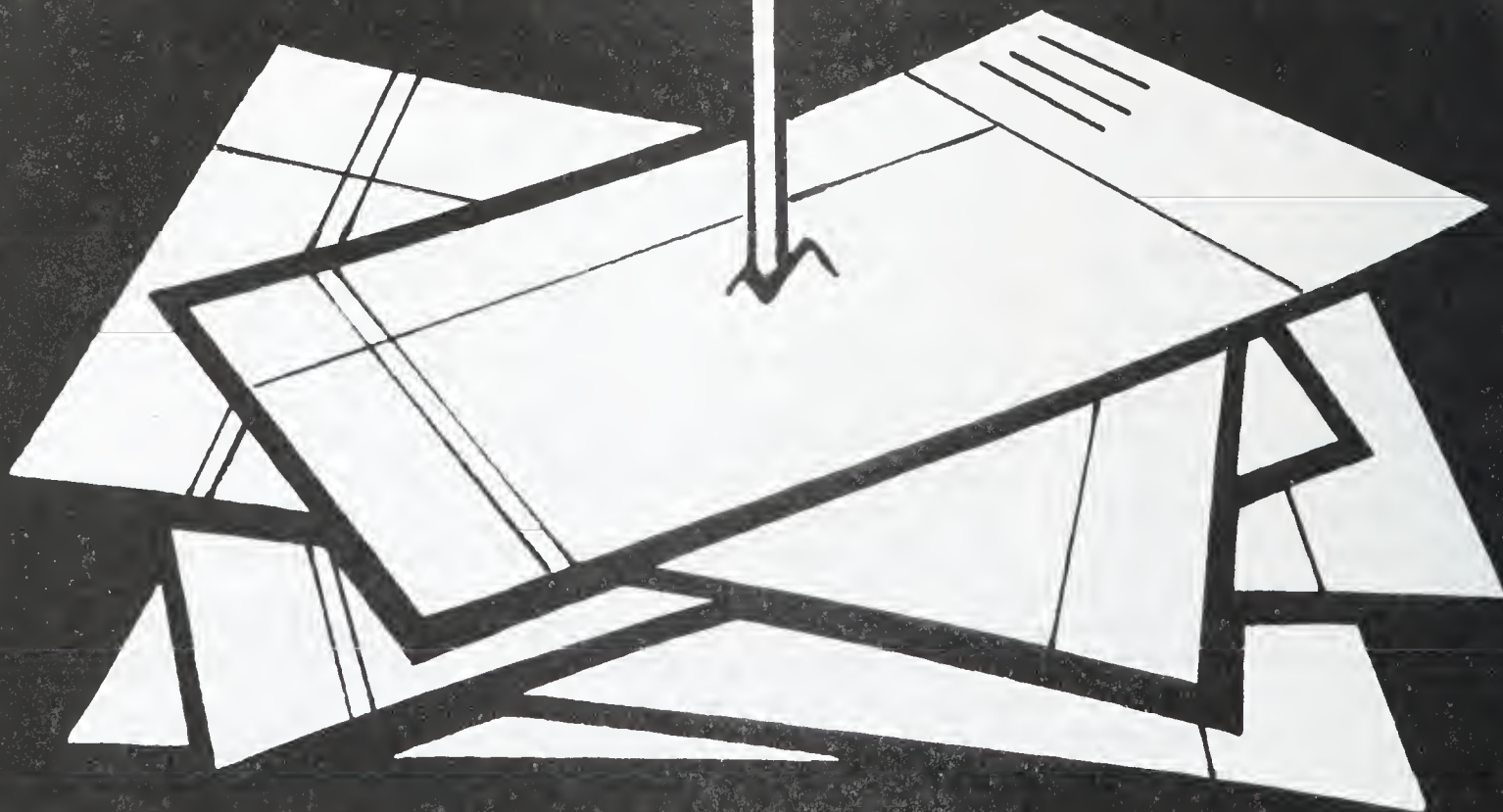
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Since 1971, the use of acupuncture analgesia as a substitute for anesthetic drugs in major surgical operations has aroused global interest. Research in this area has produced some encouraging results.

Electrophysiological studies by Pomeranz *et al.* on anesthetized cats proved that electroacupuncture reduced responses in spinal cord neurons to noxious stimuli. These inhibitions were blocked by decerebration or administration of naloxone. Furthermore, hypophysectomy blocked electroacupuncture analgesia (EAA) in mice. Clinical trials carried out by Mayer *et al.* also showed that EAA in human subjects were reversed by naloxone. Hence it appeared that EAA might be mediated via a neural mechanism which stimulated the

release of pituitary endorphins to bind to opiate receptors and produce analgesia. Further evidence and the present understanding of the endorphin system have constituted a framework in which acupuncture analgesia can postulated to act on three levels, Fig. 1:

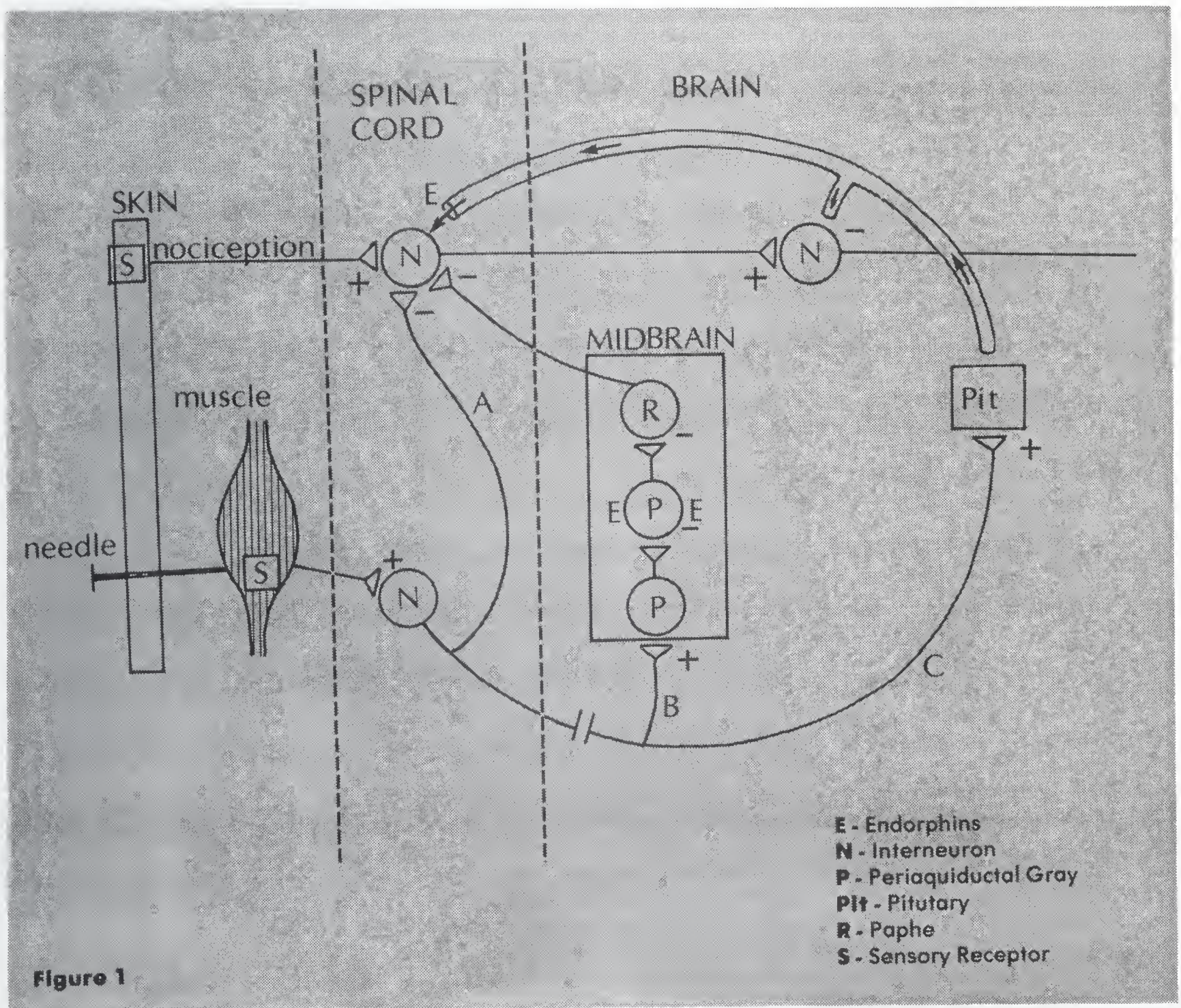
Local: Segmental release of enkephalin in the spinal cord may result subsequent to acupuncture needling. This leads to inhibition of noxious input at the dorsal horn and results in analgesia.

Regional: Acupuncture may activate the midbrain periaqueductal-raphe system, probably through a neural mechanism. Since there is a regional mapping in the midbrain, selective stimulations by appropriate afferent impulses may produce analgesia in specific regions of the

body.

General: Strong afferent stimulation may induce the pituitary to release *B*-endorphin into the blood eliciting a general analgesia.

Besides pathological pain, another area where acupuncture may be beneficial is opiate addiction. Since opiate addiction may be a result of endorphin suppression by exogenous morphine administration, acupuncture may be expected to ameliorate the withdrawal syndrome by stimulating endorphin secretion. Recent work by Pomeranz *et al.* has shown that morphine-dependent mice demonstrated suppression of opiate withdrawals after treatment with electroacupuncture. There have been some positive results from clinical trials with morphine addicts, though a well-designed controlled



study is still lacking.

Homeostasis

The high concentration of endorphins in the hypothalamus and adenohypophysis has led to investigations of their possible influence on the pituitary endocrinological functions. Both the enkephalins and *B*-endorphin were shown to stimulate growth hormone and prolactin release, while the leutinizing hormone and thyroid stimulating hormone secretions were depressed. *B*-endorphin is also a potent stimulator of vasopressin secretion.

The site of action of the endorphins seem to be at the hypothalamic level. Of greater significance is the finding that in normal animals naloxone alone produced the opposite effects hence suggesting that the endorphins may participate in regulating normal secretion of the pituitary. *B*-endorphin was also found to inhibit pancreatic secretion of somatostatin and increased insulin and glucagon release. Together with the finding of enkephalin-containing cells in the islet of Langerhans, possible endorphin influence on the endocrine pancreas is indicated.

Furthermore, met-enkephalin has been shown to depress heart rate, blood pressure and respiration when applied to the brain stem. And stimulation of the enkephalinergic neurons in the small intestine suppresses intestinal motility. All these findings seem to implicate that the endorphins may be involved in the physiological regulation of the cardio-vascular, respiratory and digestive systems.

Behaviour

The endorphinergic neurons have been shown to have extensive interactions with the acetylcholine, dopamine, GABA and noradrenaline neuronal systems in the brain. When administered *in vivo* to animals, the endorphins elicit characteristic naloxone-reversible reactions: *A*-endorphin induces tranquilization, *Y*-endorphin produces agitation and sometimes violent behaviour, whereas *B*-endorphin leads to a catatonic state. These profound behavioral effects, especially the *B*-endorphin-induced catatonia, has led to speculations that the endorphinergic system might

be involved in maintaining 'normal behaviour' and the corollary that malfunction of the system may result in psychiatric illnesses. Indeed, higher than normal CSF endorphin levels were observed in depressed or schizophrenic patients. Naloxone treatment has produced decreases in auditory hallucinations in some schizophrenics.

Moreover, like morphine, the endorphin may produce euphoria since enkephalin injections into the limbic areas caused rats to bar-press for further doses. This raises the possibility of the endorphins being the intrinsic substrate for eliciting pleasure sensations. This behavioral reinforcement behaviour pattern has also implicated the endorphins in the facilitation of long-term memory. Animal studies have also indicated a possible role of the endorphins in sexual behaviour.

Recently, it was discovered that small doses of naloxone selectively abolished overeating in genetically obese mice and rats. Elevated *B*-endorphin concentrations were found in the pituitaries of both animals. Whether the obesity is attributable to the actions of *B*-endorphin on the central nervous system, gastrointestinal opiate receptors or even the endocrine pancreas has yet to be determined. However, it could be speculated that congenital excess *B*-endorphin may be one of the etiologies of obesity in humans.

Conclusion

Pearse has advocated that all peptide-hormone-producing cells are derivatives of the neuroectoderm. Together these cells constitute a third division of the mammalian nervous system, the so-called APUD (Amine Precursor Uptake and Decarboxylation) System. The activities of this system, which subserve neuroendocrine functions, are slower in onset and of longer duration than those of the autonomic and somatic divisions of the nervous system. The restriction of the endorphins to the brain and gastrointestinal tract suggests that, like peptides such as Substance P, somatostatin, gastrin and vasoactive intestinal peptide which have similar dispositions, the endorphins may be

part of the APUD system. If this is indeed the case, the diverse effects of the endorphins may be more readily justified.

Despite its recent discovery, the endorphin system has already acquired clinical significance in several fields of medicine. Various artificial means of producing analgesia in patients suffering from chronic intractable pain such as acupuncture, transcutaneous electrical nerve stimulation (T.E.N.S.) and stimulation-produced analgesia (S.P.A.) may be mediated in part through the endorphin system. Research with the aim of producing analgesia by manipulation of the endorphins have yielded exciting results. *D*-phenylalanine, a carboxypeptidase inhibitor, has produced analgesia in animal and human studies, presumably by preventing the breakdown of endorphins at their sites of action. Of greater importance is the fact that chronic administration did not seem to elicit tolerance or physical dependence from this antipeptidase. Hence this significant finding has once again rekindled hopes for the elusive non-addictive analgesic.

Other studies in the endorphins actions have revealed genotypic differences in the opiate receptor populations and amount of endorphins in different strains. Parallels may be drawn in man to account partly for the individual variation in pain threshold and responses to opiate drugs. In addition, abnormal plasma concentrations of the endorphins are found in certain endocrine diseases thus making one wonder about the possibility of a pathological syndrome arising as a result of primary dysfunction of the endorphin system. *B*-endorphin, normally undetectable in human plasma, is found in significant amounts in plasma of patients with Nelson's syndrome, Addison's disease and Cushing's disease - endocrine disorders associated with increased ACTH and *B*-lipotropin production. The role of *B*-endorphin in the pathogenesis of these diseases is still unclear.

The profound effects of the endorphins on the body homeostasis have already been alluded to, hence

it can be appreciated that if a large amount of these peptides is released at any time into the body, life threatening conditions may result. One instance is the finding that naloxone can rapidly reverse endotoxin-induced shock. Since *B*-endorphin and ACTH are released together under all tested situations, endotoxin may therefore be expected to induce both ACTH and *B*-endorphin secretion, the latter by its potent actions on the cardiovascular system may contribute to the shock state. Therefore the role played by the endorphins in the pathogenesis of certain infectious diseases cannot be overlooked.

The possible explanation of the mechanisms of acupuncture through the endorphins has been a surprise development in the endorphin story. However, the Chinese have used acupuncture as a therapeutic treatment with creditable efficacy for an array of diseases for over 4,000 years of which analgesia is but one of its many claims. If indeed acupuncture activates the endorphin system which has extensive influence on the homeostasis of the individual it may well be able to mediate potent therapeutic effects.

An example may be the concomitant release of *B*-endorphin and ACTH, the latter induces the adrenal cortex to secrete cortisol. Both ACTH and cortisol have potent anti-inflammatory properties, which may in part, explain the efficacy of acupuncture in treating inflammatory diseases such as rheumatoid arthritis. More research is required to extend this acupuncture-endorphin hypothesis thus making acupuncture more compatible with Western medicine.

In summary, since the publication of the chemical structures of the enkephalins in December, 1975, a vast amount of new information has accumulated over a few years. A lot of questions have been answered, however, still more have been raised as a result of the wide impact of this rapidly moving field. If one accepts the present rate of progress as a gauge, future developments may well revolutionize medicine. □

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Breastfeeding: A Student's Guide Through the Milky Way

Susan Dickinson Ross 7T9

The subject of breastfeeding is one much neglected in the present day training of physicians in Canada and the U.S.. Family doctors, and even specialists in obstetrics, gynecology, and pediatrics, are all too often inadequate, if not incompetent, in the management of their potential and actual breastfeeding patients. This paper is presented with the hope of narrowing the gap between what the nursing mother needs and what the doctor provides in the way of guidance and medical treatment. As such, emphasis will be on *practical* knowledge, i.e., what you as doctor need to know.

In keeping with this emphasis, and so as not to burden you with a lengthy and redundant (not to mention boring) discourse on the anatomy and physiology of the human mammary gland (hereinafter referred to as breast), I will presume the reader has a working knowledge of such. The accompanying charts are provided only as an easy reminder, Fig. 1 & 2.

It all started with the duck-billed platypus

The monotremes (e.g., duck-billed platypuses) are the phylogenetically lowest order of animal to feed its young with milk. The young simply lick off the milk as it oozes through mother's skin. It took the marsupials (e.g., opossums) to develop the nipple. Now all milk is released at one site, convenient for the young to latch onto. The human breast is but a highly refined and

highly successful variation on this theme — highly successful, that is, until the introduction of the baby bottle in the early part of this century. The bottle was touted as liberating womankind from the tedium of breastfeeding. New 'scientific' (hence, 'better') formulae could be substituted for mother's milk. Women of the more affluent and educated classes led the way in establishing the bottle as the *modus operandi* of infant feeding for the past fifty years. Now it is generally the same class of women who lead the way in the 'back-to-basics', back-to-the-breast movement in Western society. This movement is, ironically, an outgrowth of the same Women's Lib ethos from which the bottle sprang. But now women are proudly asserting their biological femininity as well as cultural femininity more than ever before. The La Leche (lay-chay) League estimates that 38% of mothers in the U.S. start out breastfeeding their babies — more than double the number nursing 10 years ago. Lower classes generally lag by about 10 years or so in the adoption of new social modes, and, as such, one could predict a resurgence of breastfeeding in North America over the next few decades to its rightful place as the preferred way to feed our children. Unfortunately, the time lag is even greater for Third World countries, where misguided clinging to the bottle is decimating the infant population! (Kwashiorkor is the "disease of the one who was displaced from the breast".) At any

rate, as a result of the West's renewed interest in breast feeding, the doctor has the ever growing responsibility to encourage new mothers to breastfeed and to help smooth the course for those who so choose.

Milk: the beautiful food!

The Ontario dairymen's exaltation of cow's milk applies even moreso to human milk. Its benefits for both baby and mother are enormous. The milk itself is simply better for baby than any other concoction available. Its protein fraction (whey) is less than that of cow's milk (casein), but is digested with greater ease and economy of energy by baby. Colostrum is an excellent source of the 10 essential amino acids, which account for 45% of its total nitrogen content. The breastfed baby uses all the amino acids available, whereas with other milks, many amino acids are passed out in the feces unabsorbed, and malodorous as well! Constipation is very rare indeed in the breast fed baby, unlike its counterparts on cow's milk.

Total fat content is much the same, but human milk has a ratio of saturated/unsaturated fat of nearly 1.0, while cow's milk is closer to 2.0. Also, baby's gut is low in lipase, as is cow's milk, but human milk is very high in lipase. Furthermore, the lipolysis of human milk fats results in the formation of more soluble salts in the gut, the overall effect of which is a greater absorption of

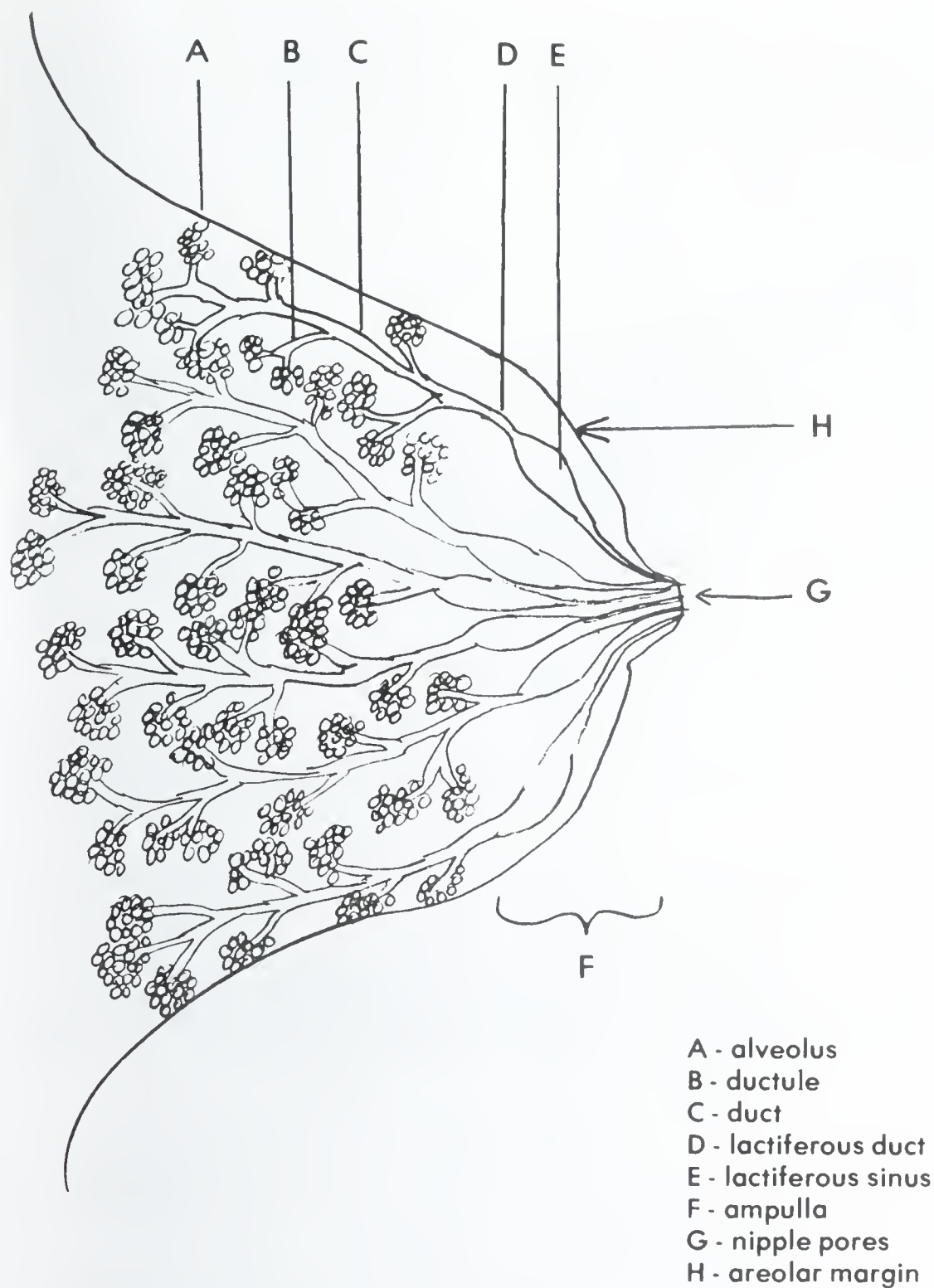


Figure 1: Schematic Mammary Gland

calcium in breast fed babies than in those fed cow's milk.

There is more carbohydrate in human milk than cow's. This carbohydrate, being lactose, as opposed to sucrose added to cow's milk formulae, encourages the growth of lactobacilli in the gut, a bacteria which suppresses the growth of pathogenic *E. Coli*, *Shigellae*, and *Salmonellae* by producing lactate and lowering the pH. (Sidelight: antibiotics in infants will knock out lactobacillus bifidus and allow more pathogenic organisms to grow. Avoid if at all possible!) Furthermore, the disaccharide lactose must be split to galactose and glucose before it can be absorbed. This results in a slow steady release of glucose into the

blood stream, in sharp contrast to sucrose, which splits rapidly into glucose and fructose and gives an early high peak of glucose in the blood stream.

The higher solute load of cow's milk taxes baby's still marginal kidney function and can cause serious metabolic imbalances when water intake is low or water demand is high (fever, diarrhea). Sodium, potassium, and calcium levels are roughly three times as high in cow's milk as in human milk, and the phosphate levels are nearly six times higher!

Other advantages of breast milk include the laxative effect of colostrum, which helps clear out the newborn's GI tract of meconium.

Another plus is complete avoidance of milk allergy, which has myriad manifestations: GI upset, skin rash, eczema, respiratory symptoms, irritability, and failure to thrive. Anemia is also less frequent in breastfed babies, even though human milk contains little iron. Many factors seem to be accountable. First, sensitivity to cow's milk protein often causes occult GI blood loss. Heat treatment of the milk tends to reduce such bleeding, but also tends to destroy other nutrients in the milk. Vitamin E, which has recently been shown to be necessary for the proper utilization of iron, is found in greater titres in human than in processed cow's milk. Deficient Vitamin E intake has been implicated as a cause of hemolytic anemia in formula fed preemies. Also, adequate levels of vitamin C, copper, and transferrin have all been reported to be necessary or helpful in the prevention of iron deficiency anemia. All exist in significantly greater quantities in human milk. Lactoferrin, an iron binding protein, is present in large amounts in human milk, but not in cow's milk. It acts to inhibit the growth of coliforms by competing for iron in the gut. (Moral: breastfed babies should **not** be given supplemental iron, at least until age of five or six months, for fear of swamping the binding properties of lactoferrin.) Finally, the high lactose and low phosphate levels of human milk promote up to 50% absorption of iron in the gut.

Other advantages of breastfeeding include less dental caries, quicker and better structural and functional development of the face and jaw and palate in particular. This has even been credited with earlier speaking in breastfed babies, with consequent earlier reading (initially a vocal activity), resulting in higher I.Q.'s! Less obesity in early childhood and later life is also suspected as resulting from breast feeding. A decreased incidence of sudden infant deaths has also been noted in infants who are breastfed as opposed to bottle fed.

Perhaps the greatest known advantage of breast milk, however, is the immunity it lends to the baby. Human milk contains IgA, IgG, IgM and IgD. IgA is by far the most important. Fully half of the

newborn's immunity is acquired via colostrum, the rest having been acquired prenatally via the placenta, mainly as IgG. The immune factors survive digestion and provide local immunity against organisms which routinely use the gut as portal of entry. These include viruses (certain strains of polio, Coxsackie, Echo, and possibly influenza), and bacteria (*staphylococcus*, *streptococcus*, *E. coli*, to which human infants are initially highly susceptible, and *Salmonella H* antigens). In addition, IgA probably prevents *E. coli*, at least, from adhering to mucosal surfaces and liberating enterotoxins responsible for diarrhea and dehydration. Furthermore, human milk contains lactoferrin (anti-coliform, see above) and lysozyme, which exerts nonspecific bacteriostatic effects.

Systemic immunity is also bolstered by breast milk because some IgA, together with IgG and IgM, is absorbed. This, along with the **exclusion** of antigenic food substances, has been credited with significantly reduced prevalence of respiratory tract illness in breast fed infants, particularly rhinorrhea,

bronchiolitis, and broncho-pneumonia. Episodes of illness, hospital admissions, and doctor visits have all been shown to be less in breastfed infants. By the age of nine to twelve months, the human infant is entirely self-supporting immunologically. This seems to be the best time to wean.

For mother, breastfeeding seems to confer protection against breast cancer, for reasons as yet unknown. The more immediate effects are the hastening of uterine involution post partum. When the newborn nurses right in the delivery room, post partum hemorrhage can be reduced by the same mechanism. It also inhibits ovulation, via high progesterone levels, due to increased prolactin and decreased FSH-LH levels. Conception is less likely to occur in the fully breastfeeding woman. Caveat: this biological contraception is **NOT** 100% effective!

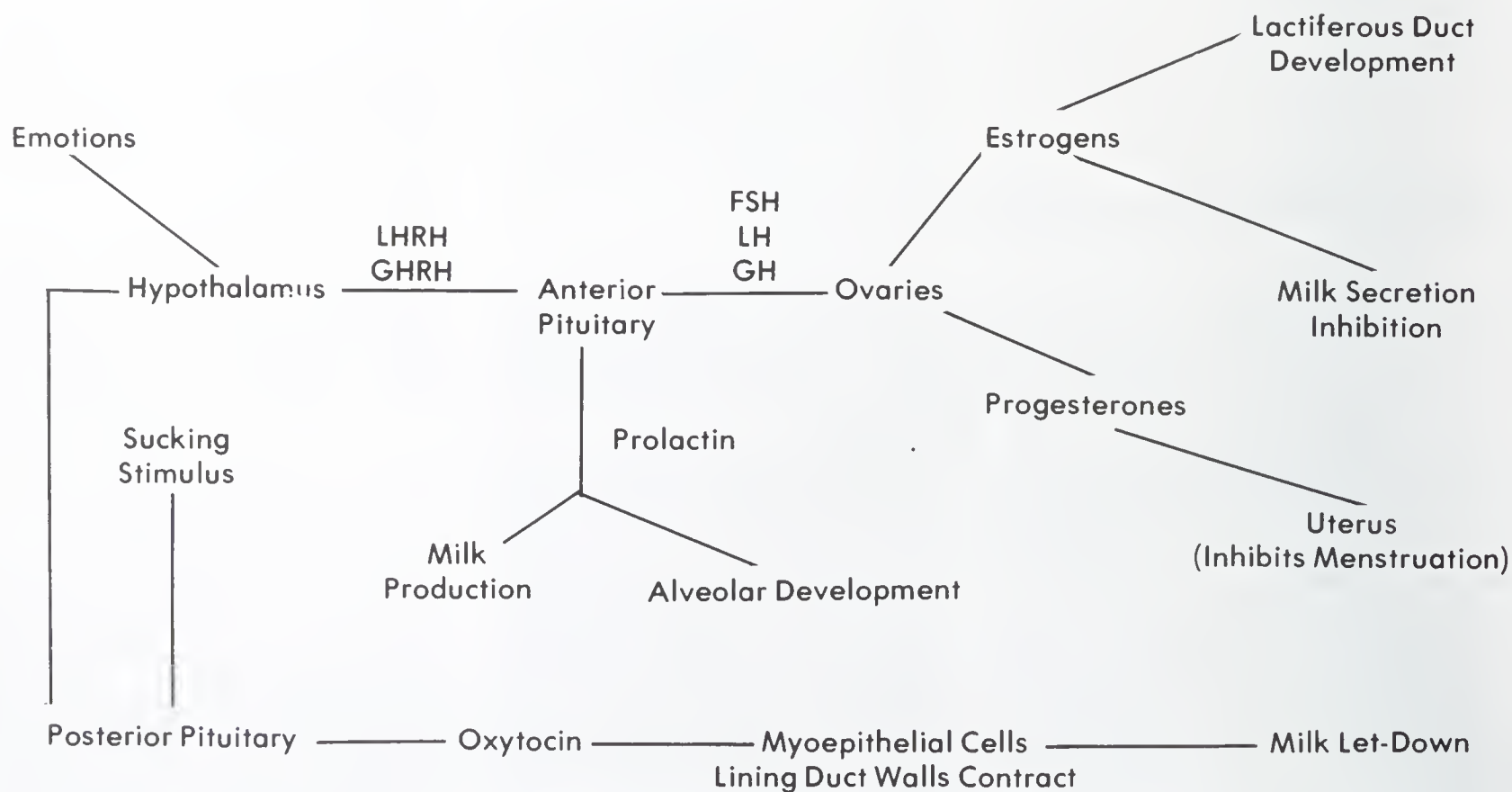
Breastfeeding has no permanent effects on the size or shape or firmness of a woman's breasts. Heredity, and the unavoidable effects of pregnancy are the major determinants of any post partum

breast changes noted. From a purely practical point of view, breastfeeding is also more convenient and less expensive. It avoids three important complications of bottle feeding: infection from poor hygiene; malnutrition from errors in preparation; and toxicity from the mistaken use of salt, for instance, in place of sugar in mixing formulae.

The intense rapport that develops between mother and baby is extremely advantageous in the psychological development of the infant, if not the mother as well. Baby's powerful sucking reflex plus his need for food are both beautifully satisfied at the breast.

Too good to be true?

Obviously breastfeeding has disadvantages too, but these are relatively insignificant in view of its aforesaid advantages. Breastfeeding cannot, of course, be delegated. Mother alone can do it. Some mothers see this as a disadvantage (usually mothers who have never known the rewards of breastfeeding!). Some researchers suspect that breast cancer viruses



Other target organs and feedback circuits exist, but only those pertinent to the breast are illustrated, due to the extreme complexity of the feedback loops.

may be transmitted in breast milk but this has never been proven. Insecticides, notably DDT, may be passed to baby in the milk, but these now-ubiquitous substances are present in cow's milk as well. Breast milk jaundice is a rare and benign entity whereby the mother secretes the steroid pregnane 3-(**A**), 20-(**B**)-diol in her milk. This steroid in the infant interferes with the hepatic conjugation of bilirubin, and thus its subsequent excretion. Such infants will appear jaundiced (seldom before the fifth day of life), but are entirely normal in all other respects, as is the mother. It disappears spontaneously by the tenth week. In the extremely rare case where serum bilirubin levels *must* be reduced, fluorescent light and/or phenobarbital for the infant are preferable to even temporary weaning.

Jaundice due to hemolytic anemia in an Rh-incompatible nursing couple is not an indication for weaning either. Although mother's anti-Rh agglutinins **do** come through in her breast milk, infants with *erythroblastosis fetalis* may be nursed safely because the antibodies in the milk diminish rapidly in the first 48 hours *and* are never significantly absorbed from the infant's GI tract.

Another 'problem' with breastfeeding is that certain drugs *must* be avoided by the mother for the duration. These include chloramphenicol, tetracycline, streptomycin, anti-thyroid drugs, cortisone, synthetic estrogens, metronidazole, imipramine, narcotics, ergot preparations, atropine, bromides, marijauna, nicotine, diuretics, anticoagulants, antimetabolites, radioactive materials, and certain oxidizing agents (see G-6-P-D list if applicable).

Problems

So, having convinced your patient to breastfeed, you must now be aware of the common problems that may arise. You must also know what to do about them!

The First Few Days:

Sleepy baby—

Be patient and do **not** supplement. Reassure mother he'll wake up and eat when he's ready. It may take up to four or five days if the

mother has had heavy analgesics prior to delivery, or a general anesthetic. Manual expression may be necessary to relieve engorgement and preserve milk production. (Have the mother cup the breast in her hand, placing her thumb above and forefinger below the nipple, at the edge of the areola. Press inward toward the chest wall, squeeze thumb and finger firmly but gently together. Do not let the fingers slide on the skin. Have her rotate her fingers to reach all the milk ducts, using about ten changes of position for each breast. She should alternate breasts every few minutes. If nothing comes at first, she will soon get the knack of it by varying the amount of inward pressure and squeeze she uses.) Also, a hand or electric pump may be used for the same purpose.

Painful mother

Sore nipples are very common, particularly in those with fair complexions. To avoid, feed **often** and on demand, certainly not on a rigid four hour schedule, which many hospitals still adhere to. Five to ten minutes on each breast will help prevent engorgement. Baby will tend to chew the nipple if his attempts to properly grasp an engorged, taut areola are futile. Also, vary nursing positions — lying down, sitting up, football hold, etc. Once a nipple has become sore, mother should begin nursing on the least sore side until the milk lets down. Warm showers and expressing some milk can help start let down as well. 'Deep cleansing breaths' and relaxation techniques often help, just as pain or emotional upset will often prevent the let down reflex. Expose sore nipples to air as much as possible. Ice packs soothe and can help the nipple protrude more, and thus make things easier for baby, and consequently for mother. UV light helps heal cracked nipples (don't get a sunburn!). Limit nursing time to 5 to 7 minutes on each side if necessary. Wear a supporting, but non-constricting bra and avoid air tight bra liners (plastic). Take a milk analgesic one half hour prior to nursing. Oxytocin nasal spray will cause milk let down, if necessary. When these instructions are followed, most cracked nipples will heal themselves within 24 hours. The Mullerian glands, as well as the

milk itself, provide the only emollients necessary. Don't wash the nipples with soap or any astringents and don't cover them with vasoline, cream, or gels. Such measures are worse than counterproductive. The only acceptable ointment is unmedicated lanolin, applied sparingly. If soreness persists, suspect thrush (Candidiasis), especially if baby develops with spots in his mouth, a persistent diaper rash, or if mother develops vaginitis. Nystatin is the treatment of choice.

Inverted nipple

Rare, and usually correctible by wearing special breast shields between nursings, and by massage. Start treatment prenatally if possible.

Later on

Mother needs 600-1000 calories extra per day. She needs more fluids as well, and **not** necessarily milk. Other sources of calcium may be utilized if the mother dislikes milk. In other words, she should eat and drink whatever she pleases, as long as it's a well balanced diet. Alcohol is permitted, in moderation, (some does get through in the milk!) and is often a boon in helping to relax mother and encourages milk let-down. Most mothers, by the way, know when they are 'letting-down' by the tight pulling feelings or 'pins and needles' they experience in the breast, this reflex will condition quite easily, usually as a response to the stimulus of baby's sucking, but also to such varied stimuli as baby's cry, changing baby's diaper, anticipating feeding baby, or even the anticipation of anything pleasurable. Leaking at inopportune moments is often the end result!

Newborns may want to eat 10 or 12 times a day at first. This is a necessary stimulus to produce more milk and mothers should give themselves fully to the 'task'. It only lasts a few weeks and is an excellent excuse to lounge around with baby and relax. By two or three months, baby will be down to six to eight feeds a day, then four to five until he's ready for a cup (age six months or so) and solid foods.

Is baby getting enough?

Not seeing exactly how many ounces baby is getting may worry

mother, especially if baby spits up a lot. If he's reasonably content and gaining weight (a pound a month is acceptable) and has lots of wet diapers, he's getting enough. Remember that babies get 90% of the milk in the first five minutes. Extra water is not necessary.

There will be times when baby seems to want to eat constantly, especially at around six weeks and three months...growth spurts. More nursing stimulates more milk production; the breast is a beautiful cybernetic unit!

Bowel movements will be *highly* variable; in color, from green to yellow to brown; in consistency, from curdy solids to just a fluid stain on the diaper; in frequency, from one per meal to one per week. The odor is, if present at all, *very* faint and not unpleasant.

Mastitis

Breast infections are often the result of faulty let-down reflex, too long between feedings, and incomplete emptying, with consequent engorgement and stasis. Cracked nipples provide a convenient route of infection, and a worn out mother will 'allow' the infection to occur. Symptoms are a painful swelling and/or localized redness or tenderness and/or systemic symptoms, i.e., fever and malaise. What to do? Apply heat - warm compresses and showers are soothing. Loosen clothing. Rest. Above all, keep nursing. If there's no improvement after 24 hours, antibiotics may be necessary. Even with an abscess requiring surgical drainage, nursing should be continued if at all possible. Nursing in such situations has been shown to speed up healing of mother, and is not harmful to baby.

Working mothers

Working part or full time does **not** preclude breastfeeding. Giving baby the breast in the morning and evening, with bottles in between, does **not** dry up milk production. The breast adapts to produce as much as is demanded, once breastfeeding has become well established.

Sick mother

Most mundane illnesses are **not** contraindications to breastfeeding. Use discretion appropriate to the circumstances.

Fathers

Breastfeeding is doomed to fail if father does not approve. Moral support from the spouse is a necessity for success, especially in the first few weeks.

How long?

Everyone who begins breastfeeding should give it at least two weeks to establish a good working pattern. Many mothers, for a variety of excuses, give up too soon, (excuses accepted by their doctors all too readily). Nine months seems to be the optimum, but more or less is quite acceptable. Some breastfeeding is better than none at all!

Supplements

Vitamins A, C, and D are routinely given to all infants (e.g., TRIVISOL). Solids should not be started until a minimum age of four months, if at all possible.

Twins

Yes, it is possible to breastfeed twins successfully! Motivation is the key to success here. An attendant in a French orphanage last century is reported to have simultaneously maintained seven babes at the breast!

Biting

Not possible **during** feeding. Simply removing baby from the breast, with a stern "No!" will usually suffice to stop any playful biting.

Erogenous?

Breastfeeding quickly desensitizes the nipple in most women so that while pleasant, it is not sexually stimulating.

Nursing Mother's Syndrome

The tired, depressed, irritable, apprehensive mother who's losing weight is likely suffering from a combination of poor nutrition and inadequate rest. Nutrition and rest, next to motivation, are perhaps the most important ingredients to happy, healthy success. B complex may be particularly helpful here (imported

beer, Brewer's yeast, etc.).

Inform

Refer the mother to her local La Leche League; the number is in the phone book. They provide a wide variety of reading materials for mothers plus professional packs for M.D.'s. Personal help is also available, as well as group meetings.

Remember

Breastfed is best fed. □

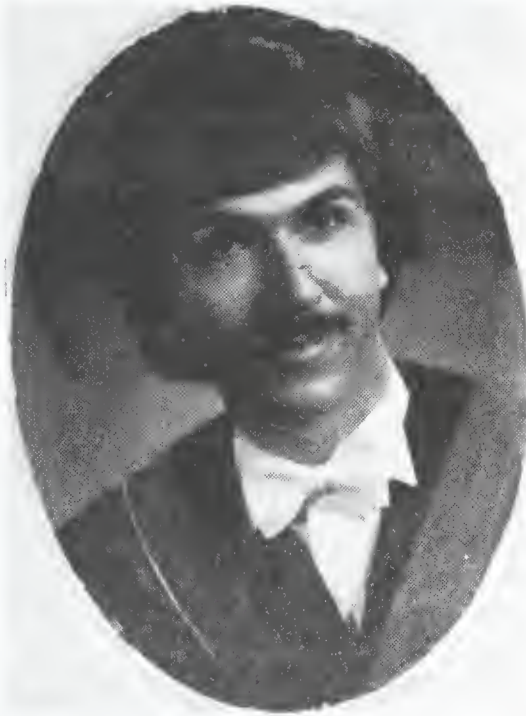
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Dr. David Naylor

Rhodes Scholar 1978

Les Urbankiewicz 8T1



Traditionally, Rhodes Scholarships are awarded to individuals who are outstanding in their chosen fields. A recent recipient, Dr. Christopher David Naylor, is clearly no exception. Dr. Naylor, a 24-year old intern at the Wellesley Hospital, graduated from the medical course at U of T in the class of 1978.

Despite his personal belief that being awarded was largely a matter of fortunate circumstances, Dr. Naylor's background exemplifies the well-rounded profile which is sought by the awards committee, who look for achievement in areas such as academic scholarship, athletics and participation in student government. During his high school days in Woodstock, Ontario, he was a professional musician. He developed a keen interest in political science and the humanities, which helped him to decide on medicine as a meaningful career. He took two years of pre-med at U of T, using the time to study social sciences, anthropology and other humanities in addition to the prerequisites.

As Dr. Naylor states, "There is a risk that in medicine, one may become a technician instead of a human biologist in general".

During the medical course, he was class president during 1974-75 and he participated in various sports including basketball and golf. He has also written several articles concerning medical and paramedical topics.

The Rhodes Scholarship will take Dr. Naylor to Oxford where he will work on a thesis toward a doctorate in philosophy (D. Phil.) for two, possibly three, years beginning this October. His area of interest is in diseases of lifestyle with particular attention to the impact of our technological and social structures on health care and health status. He notes that a crisis is rapidly

approaching because of the combination of state medicine and financial cutbacks which are placing a burden on the health care system, including teaching hospitals. While the hospitals are full of patients with diseases of lifestyle, they are forced to adjust their operation to act for the good of society as well as the individual.

Dr. Naylor's ambitions following Oxford include earning a fellowship in general internal medicine. His motives for this are twofold: first, he finds internal medicine fascinating and he feels it is important that there be good internists with a holistic approach to the patient. Secondly, he feels his credentials would give him the credibility which, regrettably, seems to be necessary to gain a teaching position in a medical faculty. He would like to lecture on the topics of social medicine and on the ethics and roles of medicine in society to both medical and non-medical undergrads.

In the interview for this article, Dr. Naylor was openly critical of the way in which doctors are trained and of medicine in general. He was deeply dismayed by the lack of good clinical skills, especially in communication, being taught in the first few undergraduate years. He feels that

these years make the deepest impression and that students need to "retain a clinical touchstone". He also feels that medical schools tend to be too technologically-oriented, turning out doctors who "spend all their time chasing the x-rays, rather than taking, or having, the time to talk to the patient". At the other extreme are the liberal medical schools which are "inviting incompetency". He advocates the teaching of medicine somewhere between these two and would acquaint students with the problem by making *The Technological Society* by Jacques Ellul mandatory reading in first year.

Of the medical field in general, Dr. Naylor notes that it has become popular to quote Lewis Thomas' notion of "half-way technology" which assumes that relentless research will eventually find inexpensive cures for everything. Naylor finds this belief "arrogant" and personally believes that society is, at present, not equipped or willing to face the implications of this research, which includes areas such as recombinant DNA. This, in Dr. Naylor's view, should be stopped or justified until society is better able to cope.

In retrospect, Dr. David Naylor may perhaps be considered as being more critical and outspoken than many previous Rhodes Scholars. However, when speaking with him, one finds him to be well-informed on the relevant issues and not willing to totally accept situations on their superficial appearance. His deep understanding of many facets of society give his criticism a constructive tone, and if Rhodes Scholars continue to be as influential in the future as they are now, then we can look forward to Dr. Naylor's contribution towards making medicine the more humanistic service to society should be. □

Gas Gangrene

Michael Ford 8T0
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Gas gangrene is commonly thought of, if it is thought of at all, as being a military problem. This is not the case. As Paul Brown¹ found, there were more cases of gas gangrene in the greater Miami area, in a comparable time, than in the whole of the Viet Nam war.

Since a hyperbaric oxygen chamber was opened in 1964, Toronto General Hospital has served as a regional referral center for a population of about 5 million. A previous study by Jackson and Waddell² of patients admitted to Toronto General helped to establish the value of hyperbaric oxygen in the treatment of gas gangrene.

The purpose of this study is to determine how matters have progressed since that initial report, with the special objectives of determining factors of importance in the prevention of gas gangrene, since for this disease, prevention is better than cure.

Materials and Methods

Clostridia are a ubiquitous, saprophytic family which cause three very lethal diseases in man: gas gangrene, tetanus, and botulism. Culture of clostridia from wounds does not automatically equate with gas gangrene, as clostridia can be cultured from about 30% of open septic wounds³. Clostridial cellulitis is defined as a self-limiting spreading infection in connective tissue with production of gas, but little toxæmia. Clostridial myositis, or gas gangrene, has been defined for the purpose of this study as infection involving

muscle with necrotic tissue, a purulent exudate and the production of gas. Another defining feature is the recovery of clostridia from the wound or a gram stain showing typical gram positive bacilli without spores.

The records of patients with gas gangrene were studied for the years 1970 to 1977. These records were considered adequate in 51 cases which exhibited the clinical features of clostridial myositis. Unfortunately, bacterial confirmation was not always present (Table I), and because it was recognized that other organisms can give a picture resembling gas gangrene, these cases were excluded, leaving 38 cases.

Clinical Features

The most common sign, inordinate pain, was present in all cases. In 69 per cent of cases fever was present, subcutaneous crepitus in 63 percent and wound discharge in 53

percent. Shock and jaundice, indicating advanced disease, were present in 41 per cent and 12 per cent respectively. Several patients had concomitant diseases, such as diabetes or cancer, which either led to diminished peripheral vascular supply or to decreased immunity.

Treatment

Gas gangrene occurs when clostridia, particularly *clostridium welchii*, gain access to tissues and are able to multiply. Gas gangrene-producing clostridia elaborate a variety of exotoxins, the most lethal being the alpha toxin produced by *clostridium welchii*, which has marked necrotizing and haemolytic properties. The organisms invade and destroy neighbouring healthy muscles. During the metabolic transformation of necrotic muscle gas is produced (Fig. 1). Toxaemia is severe and there may be haemolytic anaemia and jaundice. Bacteremia is

Table 1
Organisms Cultured

Organisms Cultured	Number of Patients
Clostridium	28 - 54%
Mixed (Clostridium & Others)	15 - 29%
Pseudomonas	2 - 4%
E. Coli	1 - 2%
Gram Positive Cocci	1 - 2%
Acinetobacter	1 - 2%
Antratus	
No Organism Isolated	3 - 6%

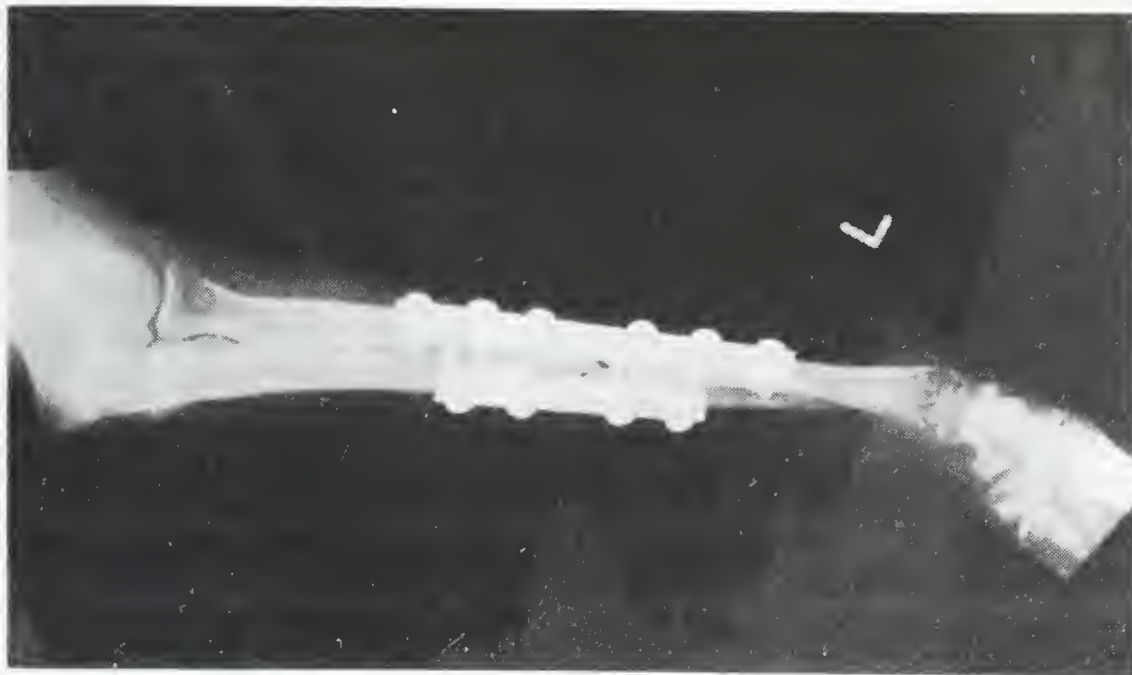


Figure 1: Gas can be seen in the volar muscles of this arm which developed gas gangrene.

common and soon results in death.

Treatment of gas gangrene consists of surgical excision of all dead and devitalized tissue, antibacterial therapy to destroy the clostridial organism and hyperbaric oxygen. The antibiotics of choice are penicillin, clindamycin or chloramphenicol⁴. Anti-gas gangrene serum is probably of no value⁵ and is no longer used in Toronto General.

Hyperbaric oxygen was introduced clinically in the treatment of gas gangrene by Brummelkamp in 1961⁶. Local applications of oxygenating substances had been tried previously as had insoufflation of the tissues with oxygen under pressure⁷. The aim was to increase the oxygen levels at the site of the clostridia; however, it was the hyperbaric chamber which made this a practical reality. The underlying physiological concept was described by Haldane in 1895⁸. Most of the oxygen in the blood is bound to haemoglobin. A small amount, normally less than 0.3 volumes per cent, is carried in simple solution. This can be increased to four volumes per cent by exposure to pure oxygen at a pressure of three atmospheres, thus allowing oxygen to diffuse from the plasma into relatively ischaemic tissues.

In 1878 Paul Bert⁹ showed that exposure to high pressure oxygen inhibited the growth of anaerobic organisms. In 1965 van Unnick¹⁰ showed that the alpha toxin production could be suppressed by oxygen at three atmospheres pressure. Once

toxin production is halted tissue death stops, the residual toxin is rapidly fixed by the tissues, and there is rapid clinical improvement.

There are certain dangers associated with the use of pressures above atmospheric:

Barotrauma

Barotrauma can result from pressure change in the gas filled cavities of the body. In practice the only problem is with the middle ear. This can usually be avoided by measures to open the Eustachian canal, such as swallowing. For those too young or too ill to cooperate myringotomy is necessary.

Dangers Associated With Compressed Air

These comprise various types of decompression sickness ranging from joint pains (type I bends) to a neurological crisis (type II bends) due to the formation of nitrogen bubbles in the neural tissue, which can result in permanent neurological damage. Avascular necrosis of bone is a further late hazard of the use of compressed air. The relationship between avascular necrosis and joint pain is not clear.

Oxygen Poisoning

High oxygen pressures cause inactivation of the sulphhydryl group of enzymes and co-enzymes in the Krebs cycle¹¹. Clinical manifestations, which may be seen at three atmospheres pressure, include convulsions preceded by autonomic and behavioural disturbances. These effects can be controlled by sedation.

Fire Hazard

Oxygen supports combustion and this is enhanced when it is under pressure. The dangers were all too clearly demonstrated by the Apollo spacecraft disaster. Strict precautions have to be taken to avoid sparking from electrical equipment and to reduce static electrical charges.

The hyperbaric therapy routine used in Toronto General Hospital consists of "dives" at three atmospheres pressure for 90 minutes. These are repeated as many as five times depending on the clinical situation. Frequently, a dramatic improvement is noted with the first dive.

Results

The 38 cases were broken down into four groups: those following trauma, amputation for peripheral vascular disease, general surgical and gynaecological procedures, and a miscellaneous group.

The thirteen cases following abdominal procedures showed a not unexpectedly high mortality rate. Eight patients died, that is, a mortality rate of 61%. Of those who died, five received hyperbaric oxygen. No useful statistical analysis can be performed on the effectiveness of hyperbaric oxygen as the numbers are too small and many of these patients were already moribund, one dying within 5 minutes of admission to hospital. As clostridia are frequently present in human faeces, gas gangrene must be looked upon to some extent as a natural hazard of bowel surgery. What was found disturbing was the relative failure to use the facilities of the hyperbaric oxygen chamber.

Gas gangrene occurred after below-knee amputation for peripheral vascular disease in 8 cases. With one exception, all cases underwent subsequent further amputation above the knee. One patient died, resulting in mortality rate of 12.5%. Hyperbaric oxygen was used in 6 cases, including the patient who died. In amputation for peripheral vascular disease an attempt is made to preserve the knee joint in virtually all patients, the consequences being that the amputation is frequently carried out through

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barely viable tissue and the skin is closed primarily in virtually all cases. since secondary closure also poses significant wound healing problems, the occasional occurrence of gas gangrene is not unexpected, and must be accepted as a natural hazard. It is our practice in Toronto General Hospital to use penicillin prophylactically in amputations for peripheral vascular disease. This would appear to be of value since the majority of cases which developed gas gangrene had not been on prophylactic antibiotics.

Of the four patients in the miscellaneous group, two followed limb lacerations which were sutured primarily. The other two developed from limb ulcers. One patient who died was 82 years old and moribund on admission, but the others recovered following debridement and grafting. Three patients, including the one who died, had hyperbaric oxygen.

Thirteen cases followed compound fractures, ten being of the lower limb and three of the upper. It is of significance that no case within this group came from another teaching hospital and every case was treated with primary wound closure, usually with internal fixation of the fracture. All but two had hyperbaric oxygen and all survived. Eight, or 61 per cent, required amputation. These figures show that there has been a significant advance in the treatment of this dreaded complication since Jackson and Waddell's study², in which a similar group had a mortality rate of 27 per cent.

Discussion

Clostridia are free-living saprophytes and are strict anaerobes. The most significant factor which allows them to grow in human tissue

is a lowering of the oxidation-reduction, or redox, potential of tissues below the normal + 0.12 volts¹².

The redox potential may be reduced by:

1. Impairment of local vascular supply by trauma to vessels, pressure by tourniquets, plaster casts or dressings, or the effect of cold, shock or local oedema.
2. Presence of foreign bodies such as clothing, metal, sutures and dust.
3. Destruction of tissue by trauma, infection, or injection of medications such as adrenaline. The presence of ionized calcium salts commonly found in fertile soil is a powerful cause of tissue necrosis.
4. Growth of aerobic organisms in a wound.

Translated into clinical terms the most significant factors with respect to post-traumatic gas gangrene are inadequate debridement and the use of primary wound closure. Gas gangrene following abdominal procedures and amputation for peripheral vascular disease must to some extent be looked upon as a natural hazard, but post-traumatic gas gangrene is a preventable disease.

Debridement is a loosely applied term, and is all too often loosely carried out¹. The significance of primary wound closure is well known to military surgeons. It used to be said that the lessons learned by surgeons in one war had to be relearned in the next. In Viet Nam the front line surgeons were issued with strict instructions that wounds sustained in the field had to be left open. This was necessitated by the fact that the front line surgeon was frequently not trained in the treatment of trauma. The results of this directive were substantial. Viet Nam

produced fewer cases of gas gangrene than any other war in history¹.

It is significant that every case of gas gangrene in this series following a compound fracture had the wound closed primarily. Hampton's¹³ statement that "for clostridial myositis which develops in a primarily closed wound there is no excuse at all" is, rather extreme, but is a statement worth bearing in mind.

Summary

This study has shown that post traumatic gas gangrene is a treatable condition, usually necessitating amputation, but it is also to some extent a preventable complication through attention to well-known and well-accepted surgical techniques, Table II. □

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Table 2

Preventative Measures

1. Recognition of wound factors such as type and degree of tissue damage and degree of contamination.
2. Adequate and thorough debridement.
3. Delayed closure of wounds in which contamination or tissue damage is great.

Child Abuse

Kirby Hsu 7T9

The public has been taking an increasing interest in child abuse in recent years. Because of denial, ignorance, lack of skills or a combination, many doctors find it difficult to deal with these cases.

Let's say you have a case of suspected child abuse in your office. What questions would you ask? What would you look for? What tests would you order? What would you say to the mother? And who would you call?

The worst you can do is to turn your back and reassure the mother that it was just an accident. Not only is it an offense not to report a case of child abuse, but the physician can be sued for damages for not reporting a suspected case of child abuse or for not taking proper actions to ensure the safety of the child.

History

Child abuse has been recorded for many centuries and in many different cultures. The Greeks could dispose of their unwanted babies; children were killed by ritual sacrifice to gods in China and India; Romans could sell, abandon and sacrifice their children as they pleased. During the crusades, infant abandonment was common; the Hospital of the Holy Ghost and the Order of the Holy Ghost were devoted to the care of these children. With the age of industrialisation, children were put to work in factories. It was considered a good thing for children to be sharing the work of the family. Children over the age of five were worked up to sixteen hours a day,

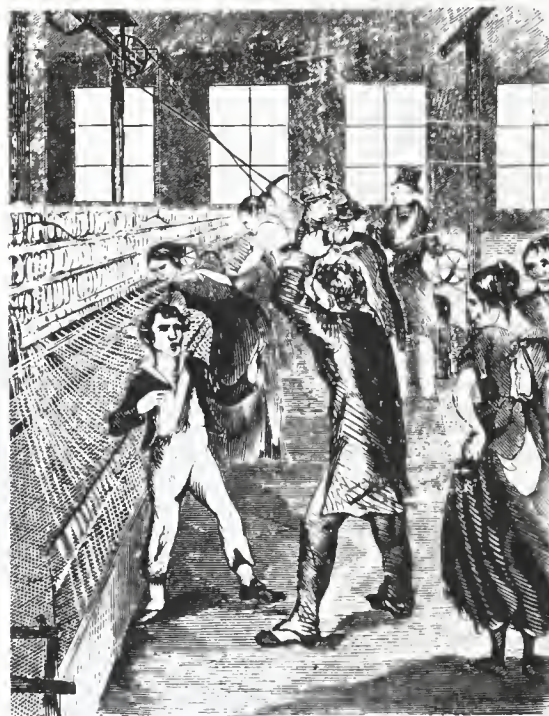


Figure 1: Child beating in an English woolen mill, around 1850.

sometimes with iron shackles around their ankles to prevent escape.

Physical punishment has always been considered necessary to maintain discipline. Five thousand years ago, in the school of Sumer, boys were punished by "the man in charge of the whip". It is written in the Bible that "spare the rod and spoil the child". In 1646, Massachusetts courts imposed the death penalty on unruly children.

Nonetheless, there were people who spoke out against child abuse. In the sixteenth century, Roger Ascham advocated love instead of fear in teaching children. The legal regulation against corporal punishment in school was pioneered at the end of the eighteenth century.

In 1802, as a reform of child labor, the first factory act was passed by parliament.

In 1871, church workers were unable to convince local authorities to take legal actions against the adoptive parents of Mary Ellen, even though she was being regularly beaten and seriously malnourished. Instead, the case was taken to the Society for the Prevention of Cruelty to Animals, who removed Mary Ellen from her home. As a direct result of this incident, the Society for the Prevention of Cruelty to Children was founded in New York City that year. Other similar societies were subsequently set up in cities across North America and Europe.

Clinical breakthrough leading to the description of the "battered child syndrome" came in 1946 when Caffey described the association of chronic subdural hematoma and fractures of long bones. As he could not obtain any history of violence in those cases, no conclusion was made.

In 1953, Astley in England described six babies with similar metaphyseal discontinuity, but he attributed them to "metaphyseal fragility". In that same year, Silverman presented three cases of unrecognized skeletal trauma which were not suspected clinically. He urged clinicians to be more thorough in obtaining a history of injury rather than in pursuing other etiologies. Two years later, Woolley and Evans gave convincing evidence that when these infants were removed from their home environment, their

lesions healed promptly and no new lesions developed. They concluded that these skeletal lesions were due to "undesirable vectors of force" in the home environment such as an unstable household, neurotic or psychotic parents.

The term "battered child syndrome" was first coined by Kempe in 1962 to draw the attention of pediatricians to the seriousness of the problem. This has resulted in significant interest and activity in not only the medical profession but also in social agencies, the legal profession, and the legislatures.

Definition

"Battered child syndrome" was originally described as a clinical condition in young children who have received serious physical abuse, generally from a parent or foster parent. In the annual meeting of the National Society for the Prevention of Cruelty to Children in London, 1968, it was pointed out that besides physical abuse, physical or mental ill health can also be inflicted by failure of parents to provide adequate needs resulting from neglect.

In a later paper, Kempe emphasized that the battered child syndrome is only "the extreme form of a whole spectrum of non-accidental injury and deprivation". At the one end, there are physical injuries from deliberate assaults, and at the other, there are emotional handicaps from passive rejections. Child abuse can therefore, be classified into five categories: physical abuse, nutritional neglect, sexual abuse, emotional abuse and neglect of medical care.

The interpretation of child abuse varies for different groups such as doctors, social workers and lawyers. Much of it depends on public opinion and the emotional climate of the localities. In recent years, a more comprehensive term - Child Abuse, Neglect, Deprivation and the Family (Child A.N.D. Family Syndrome)- is used to describe "any act of omission or commission by individuals, institutions or society and any condition resulting from such acts or inactions which deprive children of equal rights and liberties and/or interfere with their optimal development, constitute abusive or

neglectful acts which may inhibit children's development".

This expansive approach brings forward the controversial topic of rights for children -what rights should children have and how much? 1979 has been declared International Year of the Child by the United Nations and the Canadian legislature is presently considering the introduction of a separate Bill of Rights for children.

Incidence

The true incidence of child abuse in Canada or Ontario is unknown. This is due to a number of factors including the lack of standard definition of the term, the variation in reporting systems, failure to recognize cases or the reluctance to report cases.

Ten thousand to fifteen thousand children in the U.S. were severely injured by non-accidental means in 1966 and it was estimated that approximately five percent of these children were killed and twenty-five to thirty percent were permanently injured. This is statistically more significant than the problems and complications of measles, mumps and chickenpox. The incidence in Denver and New York in 1969 was found to be 225 cases per million population. The same rate has been estimated for Canada. After amendments of child welfare legislation in many States, incidences in excess of 350 cases per million were uncovered.

Using the more conservative rate of 225 cases per million population, the expected incidence in Canada would be 4,800 cases per year, and in Ontario alone it would be 2,000 cases per year. The actual number of

cases reported in Ontario isn't even half of that projection (see Table 1). In a 1972 Toronto Hospital for Sick Children report, there were 451 cases diagnosed in five years which gives an average of ninety cases per year. While the number of reported cases has been increasing in recent years, the true incidence may, in fact, be remaining constant. This is due to the increasing recognition and reporting of cases which may have been missed in the past.

Demographic Considerations

Child abuse has been demonstrated to exist in all segments of society and is not limited by race, religion, age, education, nor socio-economic status. In addition, studies indicate that the majority of child abuse occurs within two-parent families. Father and mother are equally likely to be the perpetrator of abuse and both male and female are equally likely to be the victim of abuse. Even though earlier hospital-based studies suggested that most abused children were under the age of four, later studies demonstrated a much wider range of ages (see Table 2). Most parents who abuse their children do not fall within any standard psychiatric diagnostic classification: their incidence of psychiatric illness is similar to that of the general population.

Sequelae

As more studies on child abuse have been generated in recent years, it is alarming to learn about what can happen to these children. The mortality rate of child abuse has been reported to be up to twenty-seven percent. Although it has been pointed out that the abused child can be of a wide age range, the majority of fatal cases, occur in children under the age of three. If no intervention is instituted and the child returns home, there is a twenty-five to fifty percent risk of serious permanent injury or death.

In follow-up studies of abused children, they showed delayed or retarded development physically, socially, and intellectually. Children with a history of trauma are more likely to be retarded, and even with known brain damage eliminated, IQ scores have been found to be significantly related to environmental

Table 1

Reported Child Abuse Cases in Ontario

1966.....	225
1967.....	380
1968.....	269
1969.....	370
1970.....	407
1971.....	422
1972.....	491
1973.....	598
1974.....	562
1975.....	769

Table 2
Child's Sex and Age at Time of Abuse

Sex	Age				Total	Percentage
	0-2	3-5	6-12	Over 13		
Male	166	123	191	47	527	48.3
Female	147	119	174	122	562	51.6
Total	313	242	365	169		

influences. In the most recent controlled study of fifty-nine physically abused children, a significantly higher incidence of self-destructive behaviour including suicide attempts and self-mutilation was demonstrated. In addition, these children also showed ego function deficits and impaired impulse control.

Legal Considerations

The law in Ontario relating to child neglect is contained in the Criminal Code of Canada and the Child Welfare Act. The relevant sections of the Criminal Code are penal in nature; they are intended to punish the parents for crimes committed towards the child and they require proof beyond a reasonable doubt. The Child Welfare Act is concerned solely with determining what is in the best interest of the child. It is not a punitive statute and the degree of proof required depends on the balance of probability.

Section 20(1)(6) of the Child Welfare Act deals with the definition of a child in need of protection and it encompasses far more than physical abuse as it includes emotional abuse and neglect. Another notable section in the Child Welfare Act is Section 41(1). This Section requires any individual with information of child abuse to report that information to a Children's Aid Society or Crown Attorney. A change in the Act is presently under consideration which would require even suspected cases of abuse to be reported.

There may be a feeling of reluctance in doctors to get involved in court proceedings and a belief that their time can be spent other than

making reports or attending courts. This points out the importance of making concise reports in a language understandable to the court. The legal system recognizes the value of a doctor's time and the Ontario Evidence Act provides that in many cases, a doctor's evidence can be given by way of a written report.

Etiology

Numerous factors have been proposed as the prime cause of child abuse; there is no general agreement and various factors all have a role to play. An understanding of the etiology of child abuse is not only necessary in order to appreciate the signs and symptoms of the syndrome, but facilitates the management of the cases. Basically there are five components which lead to abuse:

- 1) stimulus
- 2) parental reaction
- 3) parental preparation
- 4) precipitating factor
- 5) cultural tolerance

1) Stimulus — this is an aspect of the child which evokes a negative response in the parent. This feature of the child may not even be real, but it is the perception held by the parent that is paramount. For example, the refusal of food by a child can be perceived by the parent as a rejection of love, whereas, in fact, the child has already eaten enough.

The stimulus can be simply that the child was an unwanted pregnancy or that the parent wanted a child of the opposite sex. Other stimuli which have been found to correlate with child abuse are prematurity, mental retardation,

physical handicaps, or congenital malformations. Most commonly, the stimulus is a behaviour. Again, the behaviour can be problematic, such as a hyperactive child or it can be problematic only as perceived by the parent, such as feelings of guilt generated by normal crying in infancy.

2) Parental reaction — these are the angry destructive feelings which are experienced by the parent and directed towards the child. Different people express anger in different ways. Physical violence, withdrawal, tears and depression can all be expressions of anger. This partially explains the different clinical manifestations which all fit within the spectrum of child abuse. The physically violent parent would attack the child and result in physical abuse, whereas the withdrawing parent would reject the child and result in his failure to thrive or injuries sustained as a result of negligence.

3) Parental preparation — this is the life experiences of the parents which shape their responses to the challenges of child-rearing. Abusing parents are often themselves abused in early childhood. The concept of "violence breeds violence" is well documented; unless the abuse pattern is interrupted, it is often self-perpetuating. Studies have found that almost all abusing parents have themselves been subjected to excessive, premature and inappropriate parental demands and criticisms. This results in low self-esteem. In addition, these abusing parents have almost invariably been involved in a situation of role reversal; this is when the child is expected to give sympathy and

support to the parent. In adulthood, these abusing parents turn to their own children to seek the love and nurture which have been denied them earlier. Thus the potential of a parent to abuse depends to a great extent on his or her own childhood.

4) Precipitating factor — any life event which alters the equilibrium of the parent-child relationship can precipitate an episode of abuse. This could be loss of job, unplanned pregnancy, marital breakdown, physical illness, and so on. The event must be viewed in relation to family function and dynamics; its importance lies in the tipping of the balance.

5) Cultural tolerance — physical punishment, even for young children, is still a socially acceptable method of correction. Van Stolk concluded that “soft-core abuse is still a way of life in North America”. It can be easily seen that the more tolerant a culture is, the more likely severe abuse will result.

Having considered the various factors contributing to child abuse, one can understand that one factor may play a more important role than the others depending on the family. There are situations where several factors are present; these can be called high-risk families and they should be recognized so that intervention can be instituted before the abuse takes place.

Diagnosis

The discrepancy between the actual and expected number of cases of child abuse clearly points out the inadequacy of diagnosis and reporting. In order to make the diagnosis of child abuse, the physician must have an open-mind with a high index of suspicion. It is important to realize that the physician may only have one chance to make the diagnosis, as many abused children are often taken to different doctors and different hospitals for each episode. At times, the signs and symptoms may not be obvious and can even be misleading.

When encountered with an injury or a burn in a child, the physician should be alerted if the history has any of the following features:

- 1) Delay by the parents in seeking advice.
- 2) Discrepancies or inconsistencies

in the history.

3) A history which is not compatible with the injury.

4) Vagueness about specific and important details.

Quite often the physician is presented with complaints other than injuries. The presenting symptom can be bedwetting, learning disability, hyperactivity, aggressive behaviour, night terrors, acquired retardation, psuedoautism, poisoning, or failure to thrive. Twenty to thirty percent of all failure to thrive cases resulted from negligence. One of the commonest prodromes of the battered child is found in a mother who brings a normal baby back again and again with non-organic complaints. It is important to point out that all these symptoms can, but not necessarily, be symptoms of child abuse or neglect. In handling these cases, the physician should pay special attention to the history of the child and the family. The five etiological factors mentioned previously should be explored with the parents. The discussion should focus on the parent's perception of the child, what kind of feelings the child generates in the parent and what it is like to be living with that child. Since most parents experience anger and destructive feelings towards their children at some time, it is important to know how the parents handle these feelings and what kind of family background they are from. Finally, find out what stresses are experienced by the family at the time and how the family is dealing with them. If a combination of these factors are present in the history, the physician should be suspicious of a high-risk family and proper intervention should be taken.

There are also features in the examination of the abused child that should alert the physician. When approached by adults, these children show a “frozen watchfulness”; they make no sounds, never smile and stand quite still. Other features such as the child who does not cry when stuck with a needle, who does not object to being separated from the parents, who lies perfectly still when being dressed and undressed, should also raise the suspicion of abuse. One part of the examination should include the observation of the

interactions between the child and the parent — role reversal is often apparent.

Finally, these are several clinical findings which include child abuse as a differential diagnosis. These are:

- failure to thrive
- perioral injuries
- fractures in children less than three years old,
- frequent injuries
- old healed fractures on X-rays,
- bizzare injuries
- advanced untreated disease
- genital or perianal trauma
- clearly demarcated burn
- subdural hematoma
- skull fractures
- ruptured viscus

Any one of the above features should alert the physician to the possibility of child abuse; the diagnosis, however is made based on the combination of history, physical examination, and laboratory findings. Not only is it important for the physician to be able to diagnose overt abuse, it is equally important to be able to recognize high-risk cases.

Management

In handling abuse cases, the attitude of the physician should be therapeutic rather than judgmental; the emphasis is on the family, their needs and difficulties. The amount and degree of intervention depends on the needs of the family and the severity of the abuse. The



Figure 2: Numerous superimposed bruises of thigh resulting from repetitive blows by stick.



Extensive subperiosteal and metaphyseal lesions. The irregularity of the periosteal envelope suggests tears in this structure.

management of high-risk cases and overt abuse will be discussed separately.

Once the possibility of non-accidental injury or the potential of abuse has been recognized, the high-risk cases should be further explored. The session with the parents should be a dialogue and not an interrogation. The discussion should centre around the etiologic factors previously mentioned. The interview should be conducted as far as possible when the subject initially comes up. In some cases, however, if the parents feel extremely anxious and antagonized, it is best to invite them back soon for a longer talk. Care must be taken to make sure that these families are not lost through missed appointments.

The goals of the interview with the high-risk family should be as follows:

1) To explore the family and determine whether the family is a high-risk case which needs further intervention.

2) To determine the difficulties and inadequacies of the family and decide on the type of intervention.

3) To gain the confidence of the parents and assist them in understanding the situation and their difficulties. When the parents feel less fearful about the physician, they will be more willing to cooperate with therapy and approach for help in the future.

4) To establish adequate follow-up for the family.

For some families, the initial interview and the acceptance of the

physician as an extended family support is sufficient intervention. For other cases, it is best to have further interviews conducted by an experienced colleague or a social worker. In any case, the initial physician should be informed and involved in the management of the family.

In handling the overt cases of abuse, the basic approach is similar to that of the high-risk case, except it must be more aggressive. To begin with, the child should be admitted to hospital. Then the following procedures should be carried out:

1) Obtain a complete history and conduct a thorough physical examination. All data should be carefully recorded.

2) Perform a coagulation screen to rule out bleeding disorders.

3) Obtain complete skeletal survey.

4) Take clinical photographs of any suspicious lesions.

5) Obtain details of previous hospital admissions or visits to emergency departments.

6) Check with community agencies for any past experience with the child or family.

7) Notify the Children's Aid Society. This is a legal obligation.

The management of these cases involves a multi-disciplinary approach, and depending on the centre, the initial physician may have to coordinate the activities of the various personnel. The physician should take sufficient time to explain to the parents that it is required by law that he report such cases and it is not necessarily an indication that the parents are culpable. At the same time, the physician can empathize with the parents' feelings of anger and frustrations and raise the possibility with them of having the child living away from home for a period of time. Some parents may welcome and feel relieved by such a suggestion.

Whatever intervention is instituted, it is essential that adequate long term follow-up be arranged as part of the management programme. This may include physicians, public health nurses, social workers, homemakers, or organizations such as Parent Aides and Parents Anonymous. In the end, it is rewarding to realize that eighty

percent of the parents can be helped and the families reunited.

Prevention

The prevention of child abuse becomes more feasible as the understanding of the syndrome is increased. Primary prevention starts with breaking up the "abusive cycle", so that when these children grow up, they will not be predisposed to be abusive parents.

Family life education and counseling for adolescents and adults in preparation for marriage can lessen disappointments from unrealistic expectations, as well as fears and frustrations. This can be reinforced by prenatal classes for parents so that they are better equipped to handle the problems of child rearing. Ready availability of contraceptives and abortion can decrease unwanted pregnancies and hence unwanted children. Furthermore, social services and the medical profession can aid in reducing stress as part of the extended family support system. Recent studies have shown that increasing parent-child bonding can decrease abuse. This can be achieved if the mother is conscious during the child's birth, holds the baby as soon as possible and separation in the neonatal period is minimized.

In many cities, there is a register of high-risk infants as recognized by the family physician, obstetrician, pediatrician or well-baby clinic nurses. These families are frequently

visited by a nurse and hence any problems arising can be dealt with and resolved before it is too far gone.

Secondary prevention is by the use of a multi-disciplinary approach. Even when abuse has occurred, many complications can be prevented by instituting appropriate interventions.

Tertiary prevention of child abuse may be achieved by the establishment of adequate long term follow-up care. It is important to point out that repeated abuses are found in one-third of the cases; only with adequate follow-up care can these repeated episodes be prevented.

Summary

The problem of child abuse has existed for thousands of years; only in recent decades has the problem aroused sufficient interest and attention to be studied. The definition of child abuse varies for different groups and different people. This confusion contributes to the difficulties in collecting statistics for child abuse. Although child abuse is not limited to one segment of society but because family stresses play a role in its etiology, a higher incidence might be expected in lower socio-economic groups. Its estimated incidence in Canada is 225 cases per million population.

The diagnosis is made with a combination of history, physical examination and laboratory investigations. In the history, special attention is paid to the perception

and expectations of the child by the parent, as well as the parents' personality, family background and any current crisis. There are also features in the physical and laboratory examination which should alert the physician to the possibility of child abuse.

In managing cases of child abuse, the physician should be honest, supportive and non-judgmental; the multi-disciplinary approach is most successful.

Almost all cases of child abuse can be prevented. Its success depends not only on the medical profession but on everyone in the community. □

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Aortic Aneurysm

Daniel Drucker 8T0

Acute aortic dissection is a medical and surgical emergency, demanding of the physician both diagnostic and therapeutic facility and skill.

The term 'dissecting aneurysm' first coined by Rene Laennec¹ in 1826 is somewhat of a misnomer since in the acute case, aneurysm formation is minimal and the pathology is really a dissecting hematoma. An early description of the pathological process was given by Morgagni² in 1760 when he wrote "that the blood forcing its way by degrees obliquely through the wall and coming out under the external coat of the artery, first by drawing it from the internal coats and then by raising it as to a large kind of ecchymosis". Since then, much has been written about the origin, clinical course and treatment of dissecting aneurysm. It is, however, only within the last twenty years that much has been accomplished in the understanding, recognition and treatment of this disease. The aim of this article is to briefly review the disease process and thereby facilitate its early recognition and appropriate management.

Pathophysiology

The pathophysiology of aortic dissection, is incompletely understood. Most dissections occur in the inner one-third to one-half of the aortic wall, a relatively avascular area between the lumen and the vasa vasorum. For years, the association between medial necrosis and aortic dissection was one which satisfied

pathologists and clinicians alike. Noting the high incidence of hypertension in these patients, Carlson et al³ compared the aortas of hypertensive patients with those on non-hypertensive controls and found the prevalence of cystic medial necrosis to be greater in the hypertensive group. However, in a subsequent study from the same institution, Murray et al⁴ could no longer find a correlation between hypertension and cystic medial necrosis.

Schlatmann et al⁵ studies a group of 100 aortas from patients who exhibited no dilation or dissection of the aorta. They found that the processes of

- 1) cystic medial necrosis
- 2) elastin fragmentation
- 3) fibrosis
- 4) medionecrosis

are consistent with the normal aging process of the aorta and that the prevalence of the above increased correspondingly with the age of the aorta. They also compared the

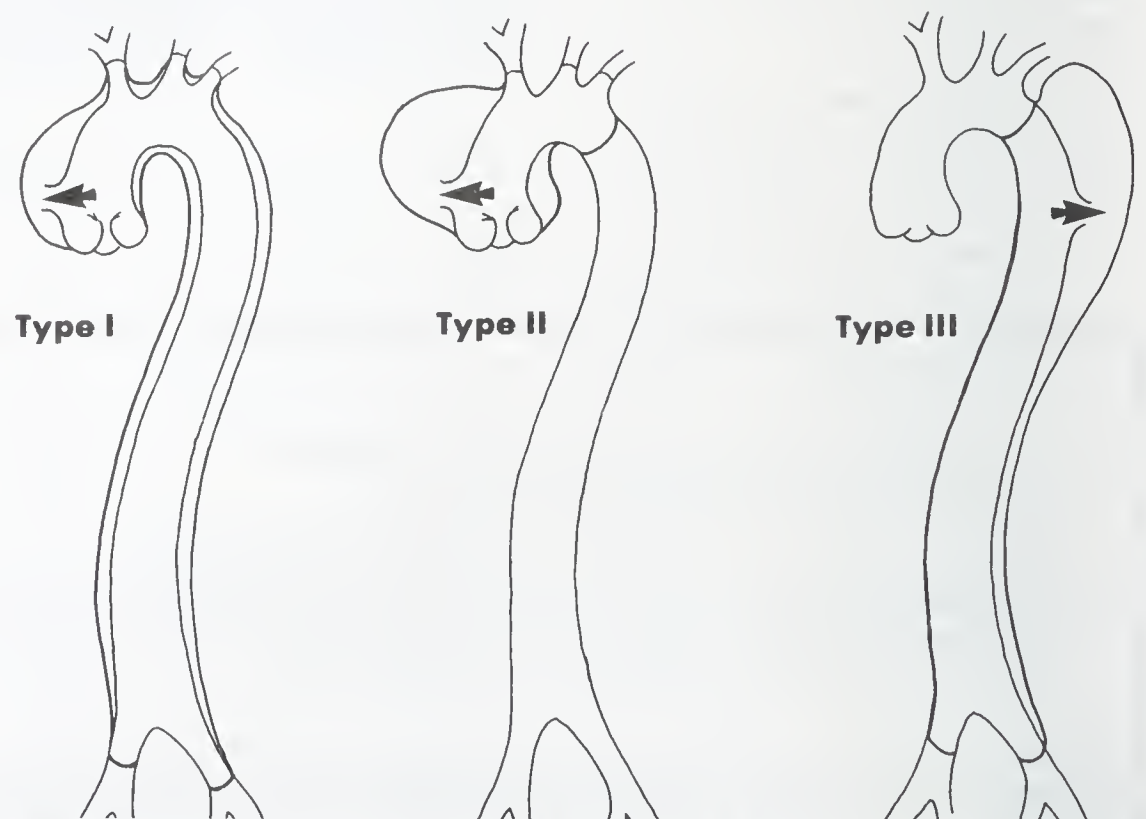
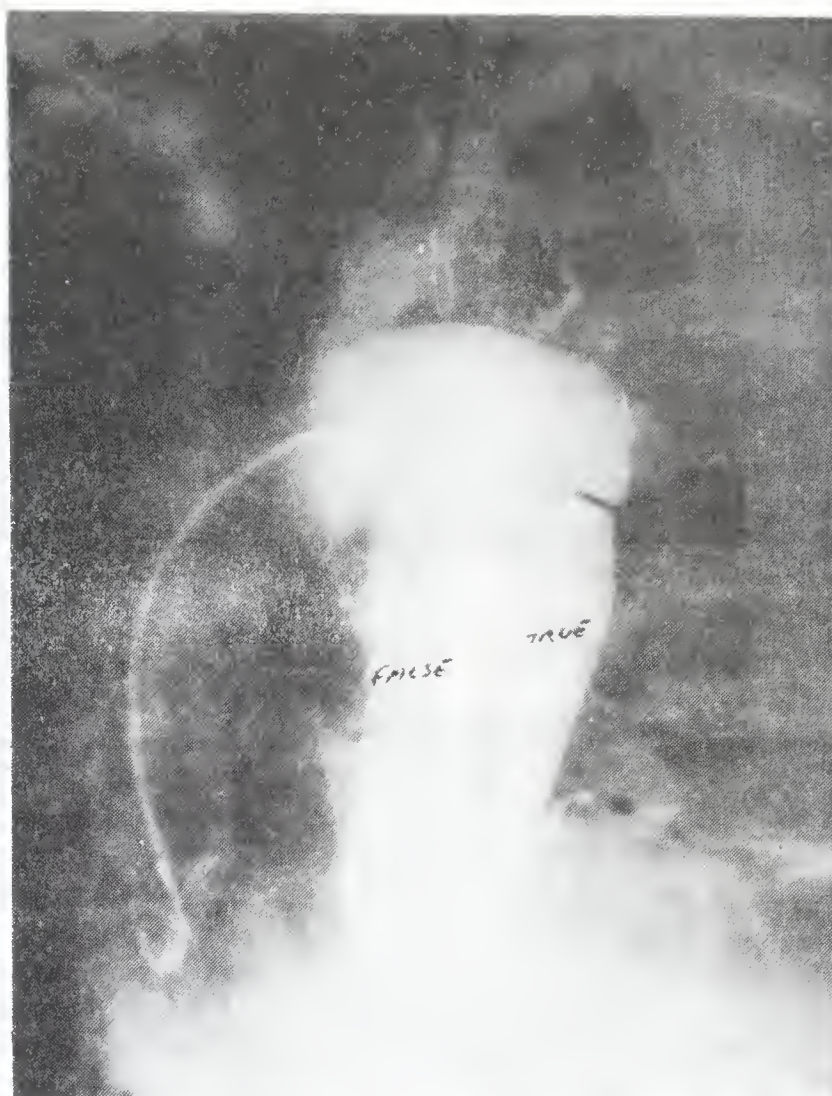


Figure 1: Classification of dissecting aneurysms of the aorta, modified after DeBakey. Type I: Dissection involves ascending aorta, aortic arch and extends distally for varying distances. Type II: Dissection limited to ascending aorta. Type III: Dissection originates at or distal to left subclavian artery and extends distally for varying distances, but does not involve aorta proximal to left subclavian artery.



Type I Aortic Dissection Aortogram: Note 1) Aortic insufficiency 2) Narrow true lumen 3) Filling of false lumen 4) Medial $\frac{1}{3}$ of aortic wall as linear radiolucency 5) Extension of dissection into neck vessels.



Type III Aortic Dissection Aortogram - Dissection begins just distal to left subclavian artery. Note true and false lumens.

prevalence of these findings in 20 hypertensive patients to 40 age-matched normotensive controls and found no significant difference.

In a subsequent study⁶ the same group compared the aortic media in patients with a dilated ascending aorta with the aortic medias of patients with complete or incomplete dissection and those with Marfan's syndrome. No qualitative differences were found between the above groups. Thus, they postulated that local circumstances determine whether a dilated aorta will rupture, or whether an incomplete or complete dissection will occur. The underlying connective tissue disorder in those patients with Marfan's syndrome simply leads to complications at an earlier age. It therefore seems that dissection is an anomaly in the normal process of injury and repair in the aging aorta and no specific medial defect can yet be held responsible for the pathogenesis of aortic dissection.

Incidence and Classification

The incidence of acute aortic dissection is 5 — 10 cases per million per year, slightly higher in the black race and more common in men than in women (2.7:1). Half of the dissections occurring in women less than 40 years of age occur during pregnancy, a markedly hypervolemic state.

It is useful to review the anatomic classification of DeBakey et al⁷ in which three types of dissecting aneurysms were identified on the basis of the site of origin and the extent of dissection (see Diagram 1). Types I and II both originate in the ascending aorta. A Type I dissection extends distal to the aortic arch, while a Type II dissection remains confined to the ascending aorta. Type III dissections begin at or just distal to the left subclavian artery and extend for varying distances into the thoracic and abdominal aortas. Type I dissections are most common followed by Type III and lastly Type

II. For the purposes of this discussion, Type I and Type II dissections may be referred to as "proximal dissections". Type III dissections as "distal dissections". The peak incidence of aortic dissection occurs in the sixth and seventh decades, the mean age of patients with proximal disease being ten years less than those with distal disease.

Symptoms and Physical Findings

The outstanding presenting complaint of patients with acute dissection is severe pain. In one well-reported series⁸, 95% of all patients presented with severe pain. It is important to query the location of the pain; anterior chest pain being more common with proximal dissection; posterior, particularly interscapular pain, more common with distal dissection. Pain was migratory in 71% of patients, the path of

Table 1

Physical Findings	Proximal Dissection	Distal Dissection
Hypertension	+	+++
Pulse Deficits	+++	+
Aortic Regurgitation Murmurs	+++	+
Neurological Deficits	++	+

Table 2

Abnormalities Seen at Aortography

Double Aortic Channel
Narrowing of the true aortic lumen
Lucent transverse intimal flap
Aortic insufficiency
Abnormally thick aortic wall
Branch vessel involvement
Abnormal catheter recoil and position

migration often corresponding to the path of vessels involved in the dissecting process. The quality of the pain has been variously described as tearing, lacerating, throbbing and ripping. Pleuritic pain often implies that the dissection involves the descending aorta. Nausea, vomiting, orthopnea and dyspnea are other symptoms frequently observed in acute dissection, hemoptysis slightly less so.

A summary of pertinent physical findings is presented in Table 1. Patients with acute aortic dissection are extremely frightened, in obvious discomfort and are often sweaty and clammy. Cardiomegaly is often present in these patients, but this is most likely due to antecedent hypertension present in most of these same patients. Up to 90% of all patients will either be hypertensive on admission to hospital or have a past history of hypertension. Pulse deficits, absence of a pulse are more common in proximal disease and involve the brachiocephalic vessels most frequently. Distal dissections have a tendency to compromise the left subclavian and femoral vessels.

Auscultation may reveal bruits over the carotid, subclavian and femoral arteries. There are frequent systolic murmurs heard over the heart and the signs of congestive heart failure may be evident, most commonly due to concomitant aortic regurgitation. A friction rub secondary to a bloody pleural effusion is sometimes audible.

Neurological deficits may include monoplegia, paraplegia, paraesthesia, hemiparesis and peripheral neuropathy. Due to the

nature of these deficits, they are often reversible. Syncope when present correlates well with occurrence of cardiac tamponade.

Diagnostic Investigations

Most laboratory investigations contribute little to the diagnosis of aortic dissection. Although two-thirds of these patients have a PMN leukocytosis, this is non-specific and of no great use. An electrocardiogram may be of great value in narrowing the differential diagnosis if myocardial infarction is suspected. Electrocardiograms in patients with acute dissection may show significant left ventricular hypertrophy secondary to antecedent hypertension, with ST and T wave abnormalities being less common. Normal electrocardiograms have been observed in up to 25% of patients with acute dissection⁸.

Most patients will have abnormal chest roentgenograms. Abnormalities that may be seen include:

- 1) widening of the superior mediastinum
- 2) abnormal aortic contour
- 3) calcium sign - separation of aortic wall intimal calcification from the outer margin of the aorta.

An associated pleural effusion, most commonly on the left side, may indicate the aneurysm is leaking. Less than 5% of patients with acute dissection will have a normal chest roentgenogram.

Aortography is extremely helpful in confirming the diagnosis and in localization and classification of the dissection. The findings at

aortography invariably influence the management of the dissection. Commonly seen abnormalities are compiled in Table 2. The presence of an intimal flap in the ascending aorta indicates a Type I or Type II lesion. Aortic insufficiency may be due to aortic dilatation and/or cusp involvement with loss of cusp support. Although previous echocardiography entailed an unsatisfactorily high rate of false-positives, Matsumoto et al¹⁰ have shown that the use of two dimensional echoaortocardiography and standard echoaortocardiography provides a more satisfactory echo diagnosis.

Management

Acute aortic dissections are managed both medically and surgically, the elective regime chosen being contingent upon the type of dissection and possible associated complications. When seeing a patient with acute dissection in the emergency room, the physician's first priority is to stabilize the patient's blood pressure. The patient should be transferred immediately to the cardiac care unit and a radial artery line for blood pressure monitoring should be set up. Aortography should not be attempted until this has been achieved. A pressure of 100-120 mm Hg systolic should be aimed for. Trimethaphan intravenously or Nitroprusside intravenously are both effective drugs for this purpose. Those electing to use Nitroprusside should be thoroughly familiar with its potential toxicity. Combinations of propranolol, methyldopa and



Dissection Involving the Abdominal Aorta - Note the presence of the left renal artery but the absence of the right renal artery, due to luminal occlusion by the dissection. The outline of the right kidney is clearly visible and the renal pelvis and right ureter may be faintly seen.

reserpine are used in addition to the above to maintain a normal pressure. The electrocardiogram, central nervous pressure and urine output should all be monitored. Once the pressure has stabilized and the pain has subsided, aortography may be performed and an attempt at aneurysm classification may be made. Many centres prefer to monitor the patient's blood pressure continuously during aortography

through a Seldinger catheter.

It is generally agreed that patients with a Type I or Type II dissection should be operated on without delay. Surgical therapy consists largely of:

- 1) resection and graft replacement
- 2) resuspension or replacement of an incompetent aortic valve
- 3) repair and end-to-end anastomoses of the aorta
- 4) local procedures to restore flow in

major branches of the aorta.

It is occasionally necessary to refer patients with Type III dissections to surgery as well. Those patients with aortic regurgitation secondary to the dissection are usually better off if operated on initially. Other indications for surgery include progression of the dissecting hematoma, compromise or occlusion of a major branch of the aorta, and myocardial tamponade or ischemia. Those patients showing signs of impending aneurysm rupture such as

- a) increase in aneurysm size
- b) blood in pleural or pericardial space
- c) inability to control pain and blood pressure

are also candidates for early surgical intervention.

Patients with Type III dissections are treated medically with an intensive drug regimen and are closely followed. Table 3 lists a few of the drugs currently used for this purpose. Some patients who have proximal dissections are not operated on. A clotted false lumen, origin of dissection not identifiable on aortogram and failure of opacification of the false channel are all contraindications to surgery. Patients with stable redissections or multiple simultaneous dissection are also usually not operated on.

Drug therapy should be continued in those patients who have undergone surgical correction and all patients should be followed and their blood pressure well controlled. Localized saccular aneurysms have developed in up to 15% of Type III patients in the follow-up period. These should be watched for and

Table 3

Commonly Used Anti-Hypertensive Medications In Aortic Dissection

Drug	Action
alpha-methyldopa	sedation, $\pm$ bradycardia, $\downarrow$ peripheral resistance
Guanethidine	bradycardia, $\downarrow$ peripheral resistance
Nitroprusside	$\downarrow$ peripheral resistance
Propranolol	bradycardia, $\uparrow$ peripheral resistance
Reserpine	sedation, bradycardia, $\downarrow$ myocardial contractility, $\downarrow$ peripheral resistance
Trimethopran	$\downarrow$ myocardial contractility, $\downarrow$ peripheral resistance

corrected surgically when necessary.

Conclusion

Recent rapid advances in cardiovascular surgical techniques and technology have contributed greatly to decreased surgical morbidity and mortality of aortic aneurysm and have helped increase both short and long term survival. Early recognition of this condition and continuously developing therapeutic modalities should further improve the outlook of patients with aortic dissection.

CASE HISTORY

Mrs. E.B., a 69 year old housewife, collapsed on the street following a sudden pain in her throat. On admission to hospital, she was comatose with a stiff neck, a left hemiplegia and bloody CSF. The working diagnosis was sub-arachnoid hemorrhage with a right hemisphere lesion secondary to a ruptured cerebral aneurysm.

She later showed a fluctuation in her level of consciousness, faint heart sounds and a changing cardiac murmur radiating into the right carotid artery. The right arm became cold and pulseless, her neck stiffness disappeared, she developed convulsions and a repeat Lumbar Puncture was normal. On the fourth hospital day, she went into shock and died.

Autopsy Findings:

- 1) Ruptured dissecting aneurysm with a) hemopericardium
b) pericardial tamponade
c) hemorrhage into mediastinum and root of lung
- 2) Extension of dissection into
a) left subclavian with partial occlusion
b) both carotid arteries with partial occlusion of left artery
c) right innominate, subclavian and carotid arteries with complete occlusion
- 3) Recent infarction of brain in distribution of right middle cerebral artery.
- 4) Bilateral bloody pleural effusions
- 5) Ascites
- 6) Herniation of cerebellar tonsils bilaterally

Cause of death: Cardiac tamponade from aneurysm rupture into the pericardial sac. □



Chest X-ray: Note widened mediastinum and separation of intimal calcification (arrow) from the outer border of the aorta. This is known as the Calcium sign.

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The author is indebted to Dr. B. Goldman for his review of this article and to Dr. R.F. Colapinto for his help in providing X-ray illustrations.

Prosthetic Valve Endocarditis

Richard Joel Friedman 8T0

With the advent of extracorporeal circulation and open heart surgical procedures, a new type of infective endocarditis has emerged with its own clinical picture and distinct microbial etiology. Infection of an artificial heart valve, called prosthetic valve endocarditis (PVE), has become a disease of medical progress, contrasted with classic primary infective endocarditis.

PVE is an infrequent but serious complication of cardiac valve replacement, associated with a high early and late mortality (approximately 56%). It was first recognized 20 years ago that foreign bodies, when placed in the cardiovascular system, predispose to infection. Geraci and co-workers in 1963 recommended prophylactic antibiotics to decrease the incidence of infections on prosthetic valves.

Over the next 10 years, as the incidence of PVE increased, the following facts emerged from various studies:

- 1) a high incidence of infection occurs surrounding the prosthesis without prophylactic antibiotics
- 2) the incidence of infection can be decreased to less than 1% when appropriate prophylactic antibiotics are used
- 3) some cases of PVE can be cured by excision of the infected prosthesis
- 4) no evidence exists that there is an increased incidence of PVE in patients who have had a prosthetic valve for the treatment of primary infective endocarditis, suggesting that early valve replacement does not increase the chances of persistent

or recurrent infection in patients when treated with antibiotics concurrently.

The use of surgery in treating PVE has increased over the years because of poor responses to medical therapy, good experience in patients requiring valve replacement for complications of primary infective endocarditis, a decreased mortality with prompt valve replacement with candidal infection and better surgical management with infected prosthetic devices.

Incidence and Etiology

The incidence of PVE varies from centre to centre and is affected by many factors, such as the patient's general condition, aseptic techniques of the surgeon, the length of time on extra-corporeal circulation, sterility of the operating room and heart-lung machine and the use of prophylactic antibiotics. In a review covering the past 10 years, the average incidence of PVE complicating cardiac valve replacement was 2%, and ranged from 1% to 9.5%. The male to female ratio was almost 3:1, and affected all ages. The aortic valve was affected almost twice as often as the mitral valve, in contrast to primary infective endocarditis where the reverse is true.

PVE is commonly divided into early or late in the patient's course, each with a different etiology and prognosis, Table 1. Early PVE, defined as occurring less than 60 days post-operative, usually occurs secondary to a septic complication of the operation, and has a mean onset

of 17 days post-operative. Some sources and predisposing factors include pneumonia, wound infection, re-operation within 48 hours, and paravalvular leaks. The mortality for early PVE is very high, ranging from 68-88%.

Late PVE, occurring more than 60 days post-op, has a mean onset of 26 months, and usually is due to an incidental septic event such as dental work, genitourinary tract procedures, or skin and wound infections. It is associated with a mortality of 38-42%.

Microbiology

Fifty-three percent of early PVE, Table 2, is caused by *Staphylococcus aureus* and *epidermidis*; including gram-negative bacilli, these microorganisms account for 75% of all instances, with consequent high mortality. Late PVE is mainly due to the streptococci, which includes *S. viridans* and the Group D streptococci (42%), although the staphylococci and gram-negative bacilli account for most of the remainder. The mortality for this group of patients is considerably lower.

In early PVE, the staphylococci, not the streptococci as in primary infective endocarditis, are the commonest and most important organisms. In primary infective endocarditis, are the commonest and most important organisms. In primary infective endocarditis, 60-80% are caused by the streptococci and only 10-30% are caused by the staphylococci. Late PVE is caused

mainly by streptococci, thereby differing from early PVE in that it more closely resembles primary infective endocarditis. The organisms are less virulent generally than those causing early PVE, accounting for the differences in mortality. This is expected due to the fact that the recognized sources of infection in the late group are not related to the surgery but rather to endogenous factors.

The gram-negative bacilli are an important group of micro-organisms causing PVE, in contrast to their low incidence in the more common forms of endocarditis. Perhaps cardiac valve prostheses are more prone to colonization by gram-negative bacilli than are natural valves.

Candida, and other fungi, have become rare but important pathogens in PVE because of their increasing incidence and almost uniformly fatal outlook (90%). First reported in 1968, it occurs in both early and late PVE. One factor concerning its increasing incidence may be the widespread use of prophylactic antibiotics predisposing the patient to an opportunistic organism such as candida.

It must be remembered that any microorganism can cause PVE. Some of the miscellaneous forms that have been reported include diptheroides, corynebacterium, klebsiella and serratia.

Table 1

PVE — Differentiating Factors Between Early And Late Onset

	early	late
onset mortality	60 days post-operative 68 - 88%	60 days post - operative 38 - 42%
common organisms	<i>S. aureus</i> and epidermis	streptococci
sources and/or predisposing factors	pneumonia wound infection reoperation within 48 hr. paravalvular leaks	dental and genitourinary procedures skin and wound infection

Pathophysiology

The presence of a foreign body nidus, in this case a prosthetic valve, encourages the growth of organisms and the production of endocardial vegetations. Propagation of these vegetations will ultimately lead to hemodynamic abnormalities.

Two processes, occurring either alone or in combination, lead to the hemodynamic abnormalities (Fig.1). Firstly, there may be mechanical failure of the poppet or disc from an encroaching vegetation, leading to a regurgitant blood flow. Secondly,

there may be direct involvement of the annular tissue, leading to disruption of the suture line of the valve at the annulus with paraprosthetic leakage.

Although the valve may be sterilized with appropriate antimicrobial therapy, the hemodynamic abnormalities will persist and result in a new regurgitant murmur, intractable congestive heart failure (CHF), and multiple systemic emboli. This is an indication for prompt placement of the infected valve.

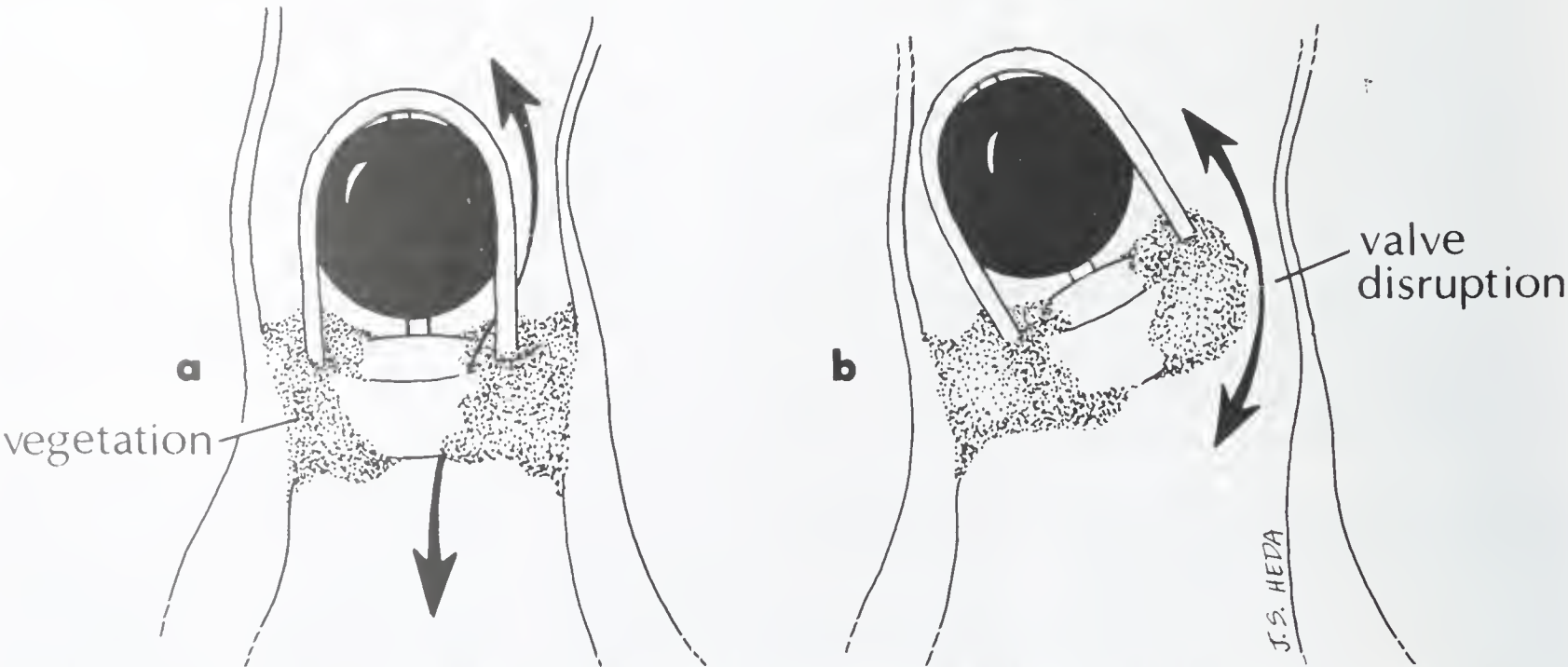


Figure 1: (a) Infected prosthetic valve with endocardial vegetation enveloping valve ring and impairing motion of the valve poppet. Arrow follows course of regurgitant blood flow. (b) Infected prosthetic valve which has been disrupted from annular coaptation as a result of destruction of endocardial integrity by organisms. Arrow follows course of regurgitant blood flow.

Table 2

Microorganisms causing PVE

organism	early PVE	late PVE
<i>S. aureus</i>	23%	11%
<i>S. epidermis</i>	30%	20%
streptococci	8%	42%
gram-negative bacilli	22%	15%
others	17%	12%

Table 3

Clinical Findings in Patients With PVE

Clinical Finding	% of Patients
fever	95
chills	70
new regurgitant murmur	49
leukocytosis ($> 12,00/mm^3$)	35
CHF	29
splenomegaly	20
peripheral emboli	17

Diagnosis

PVE may be diagnosed when 2 of the following 3 criteria occur:

- 1) at least 2 positive blood cultures with the same organism
- 2) histopathological evidence of bacterial endocarditis in surgical and/or autopsy specimens
- 3) at least 2 of the following clinical signs: fever, development of a new regurgitant murmur, newly developed splenomegaly, peripheral emboli.

Early and late PVE present with basically the same signs and symptoms, and are diagnosed the same way, the only difference being time of onset. The most common clinical findings are fever, chills and a new regurgitant murmur, Table 3. Some clinical finding tend to occur more frequently in the early or late group. A new regurgitant murmur is more commonly found in early PVE,

while peripheral emboli, including Osler's nodes, are found more often in late PVE. The reason for these differences are not known.

Treatment

Certain risk factors in PVE have been correlated with a high mortality. These include:

- 1) early onset (60 days post-operative)
- 2) high risk causative organisms (*S. aureus* and *S. epidermidis*, gram-negative bacilli, candida)
- 3) hemodynamic abnormalities (CHF, para-valvular leaks)
- 4) septic emboli
- 5) failed medical therapy (recurrent or persistent bacteremia)

It is now accepted that these high risk factors are indications for early surgical replacement of the infected prosthesis. The goals of prompt

surgical intervention are to excise the infected area before it extends into vital or inaccessible myocardial tissue, decrease the risk of embolic or vasculitic lesions, including mycotic aneurysms, and decrease the development of CHF due to valve incompetence. The risks of prompt surgical intervention include perioperative death, recurrent infection and new paravalvular leaks, yet they do not appear to be prohibitive.

With prompt surgical intervention, the patients are in a more hemodynamically stable state when operated on, therefore they can be expected to have better survival rates. The operative mortality should approach that for non-infected valve replacement in hemodynamically stable patients. In studies conducted thus far, there was a lower mortality (23%) with prompt surgical

Table 4

Medical Treatment of PVE

Organisms	Antibiotics	Alternatives
<i>S. aureus</i> or <i>S. epidermis</i>	penicillin G or methicillin	vancomycin, cephalothin or clindamycin
<i>S. viridans</i>	penicillin G and streptomycin	cephalothin or clindamycin
Group D streptococci	penicillin G and streptomycin	vancomycin and streptomycin or gentamicin
gram-negative bacilli	penicillin G and streptomycin	carbenicillin and gentamicin

Table 5
Prophylactic Antimicrobial Therapy

Procedure	Most Prevalent Organism	Prophylactic Antibiotics
dental genitourinary	<i>S. viridans</i> Group D streptococci gram-negative bacilli	penicillin G and streptomycin penicillin G and gentamicin
dermatologic cardiac catheterization	staphylococci staphylococci	cephalexin cephalexin

intervention and antibiotics compared to patients managed on antibiotics alone (60%).

The key to treatment today is to confirm the diagnosis and identify those patients at high risk for an increased mortality and replace the prosthesis without waiting for medical therapy to fail or for complications to occur. Prompt surgical replacement of the infected prosthesis is now the current treatment of choice in PVE, except for those patients in the low risk group (*vide infra*).

Antimicrobial therapy alone is used in patient falling into the low risk group not requiring prompt surgical intervention. This group includes patients with late-onset PVE, organisms associated with a low mortality (streptococci), and the absence of complications such as paravalvular leaks, CHF, emboli, and recurrent bacteremiae.

The selection of the appropriate antibiotic, Table 4, depends on indentifying the microorganism and *in vitro* susceptibility tests. Serum bactericidal levels are desirable for the greatest effect and most favourable outcome. Following discontinuation of parental antimicrobial therapy, it is recommended that blood cultures be obtained monthly for 3 months in asymptomatic patients.

Prophylaxis and Prevention

There is an important need for adequate prophylactic antibiotics in patients with prosthetic heart valves undergoing procedures that place them at an increased risk of a transient bacteremia, such as dental and genitourinary procedures, minor

skin surgery, or any operation through a contaminated area. Most instances of late PVE are probably preventable with appropriate prophylactic therapy, Table 5.

Also of concern are patients with prosthetic heart valves who acquire a localized infection in the skin or urinary tract. These patients should also receive prophylactic antimicrobial therapy. With the above measures, it is hoped that the incidence of PVE occurring in the late post-op period will decrease.

All patients undergoing valve replacement are routinely put on prophylactic antibiotics pre-, peri- and post-operatively. The efficacy of this has been recognized since the mid 1960's, but it has still not been proven conclusively from the available data that such therapy actually decreases the incidence of early PVE. Because the incidence of early PVE is low, the risk of side effects and/or complications due to the antibiotics may be greater than the risk of PVE. Therefore, the use of prophylactic antibiotics and the specific choices remain controversial.

It is obvious that preventive measures are more important than cure of an established infection in decreasing mortality from PVE, the most important being prophylactic antibiotics, management of the extracorporeal circulation and a clean operating environment. □

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The author would like to thank Dr. B.S. Goldman for review of this article.

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